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Prevalence of Thyroid Hormone Dysfunction in Patients with Hyperlipidemia; A Hospital-Based Study

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ABSTRACT

Background: Hyperlipidemia is a well-established risk factor for cardiovascular disease and may be secondary to underlying conditions such as thyroid dysfunction. Thyroid hormones influence lipid metabolism by regulating LDL receptor activity, cholesterol synthesis, and triglyceride clearance. Current guidelines recommend thyroid function screening in patients with newly diagnosed hyperlipidemia.

Methods: This hospital-based observational descriptive study was conducted at Manipal Teaching Hospital, Pokhara, Nepal. A total of 200 patients aged ≥15 years with hyperlipidemia, not on lipid-lowering therapy were enrolled. Fasting lipid profile and thyroid function tests (free T3, free T4, TSH) were measured. Associations between lipid parameters [triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), non-high-density lipoprotein cholesterol (non-HDL-C)] and thyroid status were analyzed using Pearson's Chi-square test, with p-value<0.05 considered significant.

Results: Among 200 patients, thyroid dysfunction was detected in both subclinical and overt forms. TC and non-HDL-C showed significant associations with thyroid dysfunction (p-value=0.016, φ=0.332; p-value=0.003, φ=0.428, respectively), with non-HDL-C showing the strongest correlation. TG showed a trend toward association (p-value=0.073), with overt hypothyroidism more frequent in the very high TG group (>500 mg/dL). LDL-C was not significantly associated with thyroid status (p-value=0.112).

Conclusion: Thyroid hormone dysfunction, particularly subclinical and overt hypothyroidism, is common among hyperlipidemic patients and is significantly associated with elevated TC and non-HDL-C levels. Routine thyroid function screening in newly diagnosed hyperlipidemia may facilitate early detection and management, potentially improving lipid profiles and reducing cardiovascular risk.

Keywords: hyperlipidemia, hypothyroidism; lipid profile; Non-HDL cholesterol; thyroid function.

INTRODUCTION

Elevated serum lipid levels are well-recognized risk factors for cardiovascular disease. Hyperlipidemia refers to elevated cholesterol, elevated triglycerides (TG) or both. Thyroid hormone induces the 3-hydroxy-3-methyiglutarylcoenzyme A (HMG-CoA) reductase that catalyzes the conversion of HMG-CoA to mevalonate, the first step in the hepatic de novo biosynthesis of cholesterol.[1] Thyroid hormones also activate the LDL receptors; the promoter of the LDL receptor gene contains a thyroid hormone responsive element (HRE) which allows the tri-iodothyronine(t3) to upregulate the gene expression of the LDL receptor.[2] Additionally, thyroid hormones

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stimulate the cholesteryl ester transfer protein (CETP) an enzyme which transports cholesteryl esters from HDL2 to the very low-density lipoprotein (VLDL) and triglycerides in the opposite directions.[3] Finally, thyroid hormone stimulates the lipoprotein lipase (LPL) catabolizing the triglycerides-rich lipoproteins and the hepatic lipase (HL) which hydrolyses HDL2 to HDL3.[4] Current guidelines from the national cholesterol education program the American association of clinical endocrinologists and the American thyroid association recommend screening for hypothyroidism in patients with newly diagnosed hyperlipidemia prior to starting a lipid lowering agent.[5-8] The present study is carried out to assess the association of level of lipids with thyroid hormone level and aims to find out prevalence of thyroid hormone dysfunction in patients with hyperlipidemia which might be helpful for clinical management of both the patients with hyperlipidemia as well as patients with thyroid hormone dysfunction.

METHODS

This hospital-based observational descriptive quantitative study was conducted at Manipal Teaching Hospital, Pokhara, from November 2023 to October 2024 after obtaining approval from the Institutional Review Committee. Patients from Gandaki Province who were incidentally found to have hyperlipidemia during routine outpatient visits were enrolled using non-probability sampling, provided they were aged ≥15 years, not on lipid-lowering medications, and gave informed consent; those below 15 years, already on lipid-lowering therapy, unwilling to participate, or with known thyroid disorders were excluded. Socio-demographic characteristics and co-morbidities were recorded, and fasting venous blood samples were collected after 12 hours of overnight fasting in tubes with clot activators and centrifuged at 4000 rpm for 10 minutes. The obtained serum was used to measure fasting lipid profile, blood glucose, and thyroid function (FT3, FT4, and TSH). Total cholesterol, HDL-C, and triglycerides were analyzed using a fully automated VITROS 350 dry chemistry analyzer, while LDL-C was calculated using Friedwald's equation (LDL=TC−HDL−VLDL; VLDL=TG/5). Thyroid hormones were analyzed using the fully automated VITROS ECIQ platform, and standard reference ranges were used for interpretation (TSH: 0.27-4.20 mU/L, FT4: 1.0-1.8 ng/dL, FT3: 2.3-4.0 pg/mL). Lipid categories included TG <150 (Normal), 150-199 (Borderline), 200–499 (High), >500 (Very High); Total cholesterol<200 (Normal), 201–239 (Borderline), >240 (High); LDL <100 (Good), 100–129 (Upper Normal), 130–159 (Borderline), 160–189 (High), >190 (Very High); and Non-HDL <130 (Normal), 131–159 (Borderline), 160–189 (High), >190 (Very High). All laboratory procedures followed strict internal and external quality control protocols to ensure accuracy. The minimum required sample size was calculated using the prevalence-based formula, $n=z^2pq/e^2$, taking prevalence as 9.5% and allowable error of 5%, yielding 133; however, 200 participants were ultimately enrolled to strengthen the study. Continuous variables were presented as mean ± standard deviation, categorical variables as frequency and percentage, and data were analyzed using SPSS version 20. The approval for the study was taken from the Institutional Review Committee of Nepal Manipal Teaching Hospital (MEMG/IRC/357/GA). Ethical standards were rigorously maintained, with informed consent obtained in the participants' preferred language, confidentiality assured, and full right to withdraw at any stage without any reason or consequence.

RESULTS

A total of 200 patients with hyperlipidemia were included in the study. Thyroid function status was categorized as euthyroid, subclinical hypothyroidism, overt hypothyroidism, and hyperthyroidism. The association between thyroid dysfunction and individual lipid parameters like triglycerides (TG), total cholesterol (TC), low-density lipoprotein (LDL), and non-high-density lipoprotein (non-HDL) cholesterol was assessed using Pearson's Chi-square and Fisher's exact tests. Phi coefficients were calculated to evaluate the strength of association.

Out of total 200 patients, 130 were found to have

Table 1. Thyroid status comparison between different levels of hyperlipidemia.

Lipid profile	Thyroid status					p-value	Pearson's Chi-Square value
	No.	Euthyroid	SCH	Overt hypothyroidism	Hyperthyroidism		
TG							
150-199 BL	80	74	3	1	2	0.073	11.625
200-499 H	50	46	2	2	0		
>500 VH	10	7	1	2	0		
T CHOL							
201-239 BL	88	85	2	1	0	0.016	11.327
>240 H	15	11	2	2	0		
LDL							
130-159 BL	75	71	2	1	1	0.112	10.593
160-189 H	26	23	2	1	0		
>190 VH	5	3	1	1	0		
Non-HDL							
131-159 BL	66	65	1	0	1	0.003	23.957
160-189 H	54	48	5	1	0		
>190 VH	10	6	2	2	0		

elevated TG levels, 103 had elevated T-Chol, 106 had elevated LDL and 130 had elevated non-HDL levels. Since we were analyzing hyperlipidemia, HDL levels were not considered in our study. The difference in number of patients in each group is because few patients had mixed hyperlipidemia who fall in two different categories. Triglyceride levels were found to have a moderate association with thyroid dysfunction. While the majority of patients with borderline or high TG levels were euthyroid, a higher proportion of overt hypothyroidism was observed in the very high TG group (>500 mg/dL) with a standard residual of 2.7 suggesting a statistically significant association between overt hypothyroidism and very high triglyceride level. However, the overall analysis showed no statistical significance in terms of p value though association approached statistical significance (Chi-square=11.625, p-value=0.073, phi=0.288), suggesting a possible relationship between elevated TG and overt hypothyroidism.

A statistically significant association was observed between total cholesterol levels and thyroid dysfunction (Chi-square=11.327, p-value=0.016, phi=0.332). Patients with high total cholesterol (>240 mg/dL) had a notably higher frequency of subclinical and overt hypothyroidism compared to those with borderline levels, indicating a meaningful link between elevated cholesterol and underlying thyroid hormone abnormalities. In contrast, LDL cholesterol did not show a statistically significant association with thyroid status (Chi-square=10.593, p-value=0.112, phi=0.316), although the direction of the relationship still suggested increased hypothyroidism with higher LDL levels. The absence of significance may be attributed to small subgroup sizes or overlapping lipid values across thyroid categories. The strongest association in the study was observed between non-HDL cholesterol and thyroid dysfunction. A clear rise in subclinical and overt hypothyroidism was seen in patients with high and very high non-HDL levels. The association was statistically significant (Chi-square=23.957, p-value=0.003, phi=0.428), suggesting that non-HDL cholesterol may be a useful marker for identifying patients at risk of undiagnosed thyroid dysfunction.

DISCUSSION

This hospital-based study assessed the prevalence

Table 2. Thyroid status comparison in patients with hypertriglyceridemia.

Triglyceride Level	Thyroid Status	Count	Expected Count	% Within Triglyceride Level	% Within Thyroid Status	Std. Residual
150–199	Subclinical Hypo	3	3.4	3.80%	50.00%	-0.2
	Euthyroid	74	72.6	92.50%	58.30%	0.2
	Hyperthyroid	1	1.1	1.30%	100.00%	-0.1
	Overt Hypo	2	2.9	2.50%	40.00%	-0.5
	Total	80		57.10%		
200–499	Subclinical Hypo	2	2.1	3.90%	33.30%	-0.1
	Euthyroid	46	45.4	90.20%	36.20%	0.1
	Hyperthyroid	0	0.7	0.00%	0.00%	-0.8
	Overt Hypo	3	1.8	5.90%	60.00%	0.9
	Total	51		36.40%		
≥500	Subclinical Hypo	1	0.3	12.50%	16.70%	1.2
	Euthyroid	7	7.2	87.50%	5.50%	-0.1
	Hyperthyroid	0	0.1	0.00%	0.00%	-0.4
	Overt Hypo	0	0.3	0.00%	0.00%	-1
	Total	8		5.70%		

of thyroid hormone dysfunction among patients with hyperlipidemia and evaluated the relationship between specific lipid parameters and thyroid status. The findings indicate that thyroid dysfunction, particularly subclinical and overt hypothyroidism, was relatively common among individuals with elevated lipid profiles, supporting previous evidence that hypothyroidism can contribute to lipid abnormalities through multiple metabolic pathways. In our study, total cholesterol and non-HDL cholesterol demonstrated significant associations with thyroid dysfunction, with non-HDL cholesterol showing the strongest relationship (p-value=0.003, φ=0.428). This suggests that non-HDL cholesterol, which encompasses all atherogenic lipoproteins, may be a sensitive marker for identifying underlying thyroid abnormalities in hyperlipidemic patients. Similar findings have been reported in previous studies, where hypothyroid patients often exhibited elevated non-HDL cholesterol levels due to reduced LDL receptor activity and altered lipoprotein metabolism.[10-12] Triglyceride levels showed a trend toward association with thyroid dysfunction (p-value=0.073), with overt hypothyroidism more frequent in the very high TG group (>500 mg/dL). Although statistical significance was not reached, the direction of this relationship aligns with the known role of thyroid hormones in regulating triglyceride-rich lipoprotein catabolism via lipoprotein lipase. The lack of significance may be attributable to limited subgroup sizes and potential confounding factors.[13] Interestingly, LDL cholesterol did not show a statistically significant association with thyroid status in our cohort, despite a visible increase in hypothyroid cases with higher LDL levels. This could be explained by the overlap of LDL concentrations across thyroid categories or by the relatively small number of patients in the very high LDL range, which limited statistical power. Unlike our study some other study did show some association between LDL and Thyroid hormone levels.[9] Our findings support the recommendations of major guidelines (NCEP, AACE, ATA) that advocate for thyroid function screening in newly diagnosed hyperlipidemic patients before initiating lipid-lowering therapy.[5] Detecting and treating hypothyroidism in such individuals can lead to substantial improvements in lipid parameters, potentially reducing the need for long-term pharmacological intervention and lowering cardiovascular risk.[14,15]

Limitations

Limitations of this study include its cross-sectional design, which precludes causal inference, and the hospital-based sampling, which may limit generalizability to the wider population. Additionally, exclusion of patients with secondary causes of hyperlipidemia, while necessary for reducing confounding, may underestimate the broader prevalence of thyroid dysfunction among all hyperlipidemic individuals.

CONCLUSIONS

The present study highlights a significant association between thyroid dysfunction, especially subclinical and overt hypothyroidism with elevated total cholesterol and non-HDL cholesterol levels in patients with hyperlipidemia. Non-HDL cholesterol emerged as the most strongly correlated lipid parameter, suggesting its potential utility as a screening marker for underlying thyroid abnormalities. These findings reinforce the importance of routine thyroid function testing in patients with newly diagnosed hyperlipidemia prior to starting lipid-lowering therapy. Early identification and management of hypothyroidism in such cases could improve lipid profiles, reduce cardiovascular risk, and optimize patient outcomes. Future large-scale, community-based longitudinal studies are warranted to further elucidate the causal relationships and evaluate the impact of thyroid hormone correction on lipid metabolism.

Conflict of interest: None

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