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Association between Thyroid Stimulating Hormone and Lipid Accumulation Product in Hypothyroid and Euthyroid Adults in Nepal

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ABSTRACT

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Introduction: Hypothyroidism adversely affects lipid metabolism and increases cardiovascular risk. The Lipid Accumulation Product index, based on waist circumference and triglycerides levels, is a simple marker of central lipid accumulation. Hormone deficiency in hypothyroidism leads to elevated cholesterol and triglycerides, both of which contribute to higher lipid accumulation product scores. Exploring this association may provide insight into the cardio metabolic burden of thyroid dysfunction.

Objective: To compare the Lipid Accumulation Product Index in hypothyroid and Euthyroid adults and assess its correlation with serum Thyroid Stimulating hormone level.

Methods: Cross-sectional comparative study conducted at Dhulikhel Hospital in 130 hypothyroid and 130 euthyroid adults after ethical clearance from Institutional review committee, Kathmandu University School of medical Sciences. Lipid Accumulation Product was calculated using standard gender-specific formulas. Statistical analysis was performed using Spearman correlation and logistic regression.

Results: Median Lipid Accumulation Product is higher in the hypothyroid group (54.1) compared to the euthyroid group (38.3), suggesting that hypothyroid patients tend to have greater lipid accumulation ($p < 0.001$). Thyroid Stimulating hormone was significantly associated with Lipid Accumulation Product even after adjustment for age and sex ($p < 0.05$).

Conclusion: Patients with hypothyroidism have significantly higher levels of Lipid Accumulation Product index. Thyroid Stimulating hormone and lipid accumulation product are positively correlated, supporting it as a metabolic risk marker in thyroid dysfunction.

Keywords: Cardiovascular risk; dyslipidemia; hypothyroidism; lipid accumulation product index; thyroid stimulating hormone.

INTRODUCTION

Hypothyroidism, one of the frequently encountered endocrine disorders, creates marked metabolic upheaval, chiefly through lipid alterations due to diminished thyroid hormone influence on the liver and impaired regulation of low-density lipoprotein (LDL) receptors.[1]

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Dyslipidemia in hypothyroidism, characterized by elevated LDL and triglycerides, is a major contributor to cardiovascular disease (CVD) risk worldwide.[2] South Asia is experiencing an escalating burden of thyroid disturbance, and the presence of metabolic syndrome alongside high rates of type 2 diabetes mellitus magnifies the cardio metabolic threat.[3] In Nepal, thyroid disorders often go undetected, and their metabolic and cardiovascular consequences are frequently overlooked, despite growing evidence of their link with dyslipidemia and public health burden.[4]

The lipid accumulation product based on the levels of triglycerides and waist circumference has been a useful indicator of visceral adiposity and associated with the cardio metabolic risk. It has been established as a predictor of T2DM and metabolic syndrome but has been less evaluated for the prediction of thyroid dysfunction, particularly in South Asian settings with a very high prevalence of central obesity and dyslipidemia.[5]

This study aimed to compare metabolic differences between hypothyroid and euthyroid individuals and to evaluate cardiovascular risk using TSH and the LAP index.

METHODS

It was a comparative, cross-sectional study design to study the relationship between TSH and LAP in hypothyroid and euthyroid population who visited Out Patient Department of Dhulikhel Hospital during the period of six months, May - October, 2024. Ethical approval was taken from Institutional Review committee, Kathmandu University School of Medical Sciences (IRC, KUSMS Approval no. 137/24). Even though we took consecutive sampling, the sample size was calculated as below to determine exact number of participants, where Level of confidence measure (Z) for case and control: $Z_{\alpha/2}=1.96$ for $\alpha = 0.05$, $Z_{\beta} = 0.84$ for 80% power, Margin of

error (e)= 0.25, Mean (from previous study)[6]: Mean of case(μ1)= 38.6, Mean of control(μ2)= 30.9 and Standard Deviation (σ)= 15.6 respectively.

With using the formula,

$$n = \frac{(Z\alpha/_2 + Z\beta)^2 2\sigma^2}{\mu_1 - \mu_2}$$

Considering 25% margin of error, we collected data of all male and female patients undergoing serum TSH and serum TG test in the Department of Biochemistry, Dhulikhel Hospital during. The sample size calculated was 260 participants.

For control patients referred to Department of Clinical Biochemistry of Dhulikhel Hospital with normal thyroid function test and no history of metabolic disorders. Patients aged 18 years and above who consented and fulfilled the exclusion criteria (who are on lipid lowering and anti-thyroid medications, pregnant and lactating women and having critical illness or mental illness) were included in the study. The LAP index was calculated using WC and serum TG levels, with reference values of 65 cm for males and 58 cm for females based on the original definition i.e. LAP Index: Males: (WC-65) × TG (mmol/L), Females: (WC-58) × TG (mmol/L). The data were collected in semi-structured pro forma and entered in MS-excel.

The Kolmogorov-Smirnov test was used to test for normal distributions after which we found that age, TSH, TG and LAP index were not normally distributed. Median with minimum (min), maximum (max) values were calculated for non-normal mean ± standard deviation (SD) for normally distributed variables. Statistical analysis was performed with Stata version 15. A P<0.05 was considered significant.

RESULTS

A total of 260 participants were recruited in the study, among which 130 were euthyroid and 130 were hypothyroid. The descriptive statistics characteristics of baseline parameters. (Table 1) About 86(33.08%) were male and 174 (66.92%) were female. Among the controls, 49 (37.69%) were males and 81 (62.31%) were females. Among the case, 37 (28.46%) were males and 93 (71.54%) were females. Majority of Hypothyroid participants were seen of age group 40-49 years (23.85%) whereas in Euthyroid majority were seen of age group of 20-29 years (28.46%).

Table 1: Descriptive statistics of study variables.

Variable	Median (Min, Max) / Mean (±SD)
LAP	46.48 (1.63, 346.42)
TSH (mIU/L)	3.68 (0.41, 1051.0)
TG (mmol/L)	1.65 (0.12, 7.53)
Age (years)	42.0 (18, 80)
WC (cm)	88.7 ± 10.4
Weight(kg)	64.3 ± 11.4

The study compared baseline characteristics between participants with euthyroidism and hypothyroidism. The median age was higher in the hypothyroid group (45.5 years) than in the euthyroid group (38 years). Mean weight was similar in both groups (64.23 kg vs. 64.31 kg). Mean waist circumference was slightly greater in hypothyroidism (90.12 cm) compared to euthyroidism (87.17 cm). Triglyceride levels were notably higher in hypothyroid participants compared to euthyroid participants. (Table 2)

Table 2: Mean Age, Weight and WC among Euthyroids and Hypothyroids.

Variables	Euthyroidism (Mean±SD)/ Median(min,max)	Hypothyroidism (Mean±SD) /Median(min,max)
Age (in years)	38(18, 70)	45.5(18, 80)
Weight (in kg)	64.23 (±11.92)	64.31 ±10.94)
WC (in cm)	87.17 (±10.89)	90.12 ±9.69)
TG(mmol/L)	123(39, 513)	171.5(53, 667)

Hypothyroid patients had significantly higher LAP values than euthyroid controls ($p < 0.001$). Logistic regression analysis revealed a significant association between TSH and LAP after adjusting for age and sex (OR = 1.849; 95% CI: 1.457–2.348; $p < 0.001$). (Table 3)

Table 3: Comparison of LAP among Euthyroidism and Hypothyroidism.

	Euthyroidism	Hypothyroidism	p-value
LAP Median (min,max)	38.31(1.63, 71.62)	54.10(8.30, 51.08)	<0.001*

***Significant at 0.05 by Mann Whitney U test Association between TSH and exposure variables**

Logistic regression was performed with thyroid status (hypothyroid vs. euthyroid) as the dependent variable. Predictor variables considered included age, sex, and LAP index. In multivariate analysis, after adjusting for age and sex, LAP showed a significant association with hypothyroidism (aOR =1.85, 95% CI: 1.46–2.35, $p < 0.001$). Age and sex were not significant predictors (Table 4).

Table 4. Multivariate logistic regression showing association between thyroid status and exposure variables.

Variables	Adjusted Odds Ratio (OR)	95% CI	p-value
Sex	1.52	0.90–2.55	0.115
LAP index	1.85	1.46–2.35	<0.001*
Age group	1.16	0.98–1.36	0.084

There was a positive but weak correlation between TSH and LAP (Rs=0.054, p>0.05) as given in figure 1.

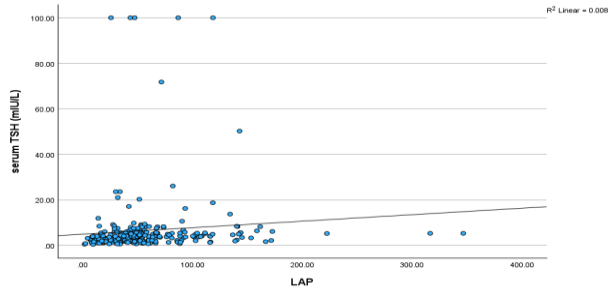

Figure 1: Correlation of LAP index with TSH level

DISCUSSION

This study aimed to assess metabolic differences between hypothyroid and euthyroid individuals and evaluate cardiovascular risk using the LAP index and TSH levels. We found that hypothyroid participants had significantly higher LAP values compared to euthyroid controls. While Spearman's correlation showed only a weak direct link between TSH and LAP, multivariate logistic regression confirmed a significant, independent association after adjusting for age and sex. These findings suggest LAP may serve as valuable early marker of metabolic stress in thyroid dysfunction.

Our results align with prior studies from Nepal and India documenting dyslipidemia and fat deposition in hypothyroid patients.[6,7] Mechanistically, reduced thyroid hormone activity impedes lipid clearance via downregulation of LDL receptors and lipoprotein lipase, promoting lipid accumulation.[1,8] Additionally, hypothyroidism triggers oxidative stress, chronic low-grade inflammation, and dysregulation of adipocytokines, all of which contribute to visceral fat accumulation and increased cardiovascular risk.[9,10] Vascular dysfunction and impaired apolipoprotein pathways particularly reduced ApoAV also exacerbate lipid disturbances in hypothyroidism.[11,12]

Global data further support the early metabolic impact of thyroid dysfunction. A Korean population based study showed that even subclinical hypothyroidism was associated with elevated LAP and heightened risk of metabolic syndrome.[13] More recent evidence indicates the broader cardio metabolic implications of thyroid-related lipid dysregulation, including associations with non-alcoholic fatty liver disease (NAFLD)[14] and cardiovascular outcomes.[15] These studies reinforce the utility of LAP and routine metabolic monitoring in hypothyroid patients, especially in resource-constrained settings.

Our study has certain limitations. Its single-center design and modest sample size may limit generalizability. We did not assess lifestyle factors such as diet, physical activity, or smoking nor did we measure inflammatory markers or insulin resistance parameters, which could enhance mechanistic understanding.

CONCLUSION

This study highlights a significant association between hypothyroidism and elevated LAP, indicating that thyroid dysfunction contributes to early metabolic disturbances and increased cardiovascular risk. Although the correlation between TSH and LAP was modest, logistic regression confirmed LAP as an independent marker of metabolic stress in hypothyroid individuals. Given its simplicity, LAP may serve as a practical screening tool in resource-limited settings for identifying individuals at higher cardiometabolic risk. Future research should involve larger, multicenter cohorts with comprehensive metabolic and vascular profiling to validate these findings and strengthen clinical applicability.

Conflict of Interest; none.

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