## Original Article

# An Influence of Clinical and Subclinical hypothyroidism on blood Lipid profile and its correlation with BMI

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## ABSTRACT

**Background:** Hypothyroidism is a common endocrine disorder worldwide, which is more prevalent in females than males. As thyroid hormones are important modulators of intermediary metabolism, any thyroid dysfunction leads to dyslipidaemia, which increases the risk of atherosclerosis. **Aims:** To compare the lipid profiles between hypothyroid groups of patients and to evaluate the correlation with body mass index (BMI). **Materials & Methods:** A Cross-sectional study using non-probability, purposive sampling was conducted from September 2024 to August 2025 at Zainul Haque Sikder Women's Medical College Hospital. Based on thyroid profile, the subjects were divided into three groups: euthyroids (n=44), subclinical hypothyroids (n=47), and clinical hypothyroids (n=42). Data were analyzed using SPSS version 18. **Results:** The mean age of patients with Clinical hypothyroidism, subclinical hypothyroidism, and Euthyroidism was 38.90 ± 10.69, 37.23 ± 12.96, and 34.95 ± 14.26 years, respectively. TSH levels increased significantly ($p < 0.05$) from 7.10 µIU/ml in patients with subclinical hypothyroidism to 17.15 µIU/ml in those with clinical hypothyroidism, compared with 2.21 µIU/ml in euthyroid individuals. FT4 levels were 0.49, 1.10, and 1.20 ng/dL in thyroid, subclinical hypothyroidism, and Euthyroidism, respectively. Mean BMI was significantly increased from 25.67 ± 4.49 for subclinical hypothyroid and 27.42 ± 4.99 for clinical hypothyroid, respectively, compared to 24.66 ± 5.67 for euthyroid. Mean Serum cholesterol was significantly ($p < 0.05$) increased from 220.98 ± 30.41 to 193.60 ± 39.47 mg/dl for clinical and subclinical hypothyroidism compared to 174.41 ± 41.11mg/dl for Euthyroid patients. Mean LDL was significantly increased from 134.43 ± 28.77, 114.53 ± 30.83, and 102.09 ± 37.82 mg/dl for clinical and subclinical patients, respectively, compared to 102.09 ± 37.82 mg/dl for Euthyroid patients. Mean Trygliceride was significantly increased from 242.21 ± 68.17, 174.74 ± 85.34, and 147.70 ± 68.63mg/dl for Clinical, Subclinical, and Euthyroid, respectively. Mean HDL levels were 36.82 ± 6.94, 41.83 ± 11.14, and 42.43 ± 8.02mg/dL among the Clinical, Subclinical, and Euthyroid groups, respectively, with no significant differences observed between hypothyroid and Euthyroid subjects. **Conclusion:** Both hypothyroid groups showed an altered lipid profile, which was significantly raised when compared with the euthyroid subjects. Such a dyslipidemic status is significant not only for managing thyroid disorders but also for common diseases such as obesity and coronary atherosclerosis in the population.

**Key Words:** Clinical hypothyroidism, Subclinical hypothyroidism, Lipid profile, TSH**.**

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## Introduction

As a common disorder, Hypothyroidism results from decreased secretion of total thyroxine (T4) and triiodothyronine (T3), leading to insufficient thyroid activity. It is more prevalent in females than in males.[1] It results from a primary process in which the thyroid gland produces insufficient thyroid hormones. Worldwide, the most common cause of hypothyroidism is iodine deficiency, with an overall prevalence of hypothyroidism reported to be 2 - 5%.[2] The estimated prevalence rates of hypothyroidism and subclinical hypothyroidism in Asia are 4.1 and 5.4%, respectively.[3] Biochemically, hyposecretion of T4 and T3 leads to a marked increase in serum thyroid-stimulating hormone (TSH) levels due to hypersecretion of pituitary TSH.4 The serum level of TSH, which is a reliable one for the biological activity of thyroid hormones, is used to diagnose clinical and subclinical hypothyroidism.[4] Thyroid dysfunction, particularly hypothyroidism, is associated with dyslipidemia as thyroid hormones are important modulators of intermediary metabolism.[5] Biochemically, clinical hypothyroidism shows a marked increase in total cholesterol (TC) and low-density lipoprotein (LDL) due to a reduced number of LDL receptors in the liver, leading to decreased fractional clearance of LDL.[6] Some studies have reported that dyslipidemia may also be accompanied by increased serum triglycerides (TG) and decreased high-density lipoproteins (HDL) in subclinical hypothyroidism.[7] Hyperlipidemia, especially hypercholesterolemia, is a significant risk factor for atherosclerosis.[8] Atherosclerosis is a complex, multifactorial disease that develops in the arterial walls due to various factors such as high lipid levels (hyperlipidemia) and high blood pressure (hypertension). The risk of developing atherosclerosis is heightened by very low-density lipoprotein (VLDL), which is a significant component of total serum cholesterol. By curing hypothyroidism, a leading cause of dyslipidemia, one can save on the possible long-term cost of cholesterol-lowering drug therapy as well as reduce the incidence of cardiovascular events.[6] The International Obesity Task Force has stated that the approach to obesity should be considered based on regional variations. Among Asians, the cut-off value for defining obesity is 23 kg/m2, with classification based on BMI values ranging from 22 to 26 kg/m2. However, the clear-cut definition of obesity is based on a BMI greater than 26 kg/m$^2$.[9] The connection between obesity, lipid profiles, and thyroid dysfunction is a significant concern for researchers. Studies are being conducted to explore the correlation among these three conditions.[10] Lipid abnormalities have also been reported in obese individuals.[7] Recently, an increasing prevalence of obesity has been observed in the urban population.[11] The objective of this study was to compare the lipid profile of the subclinical and clinical hypothyroid groups, and to assess if there is an association between obesity and lipid profile in the urban hypothyroid population of Bangladesh.

## Materials and Methods

This cross-sectional study was conducted in the Department of Medicine, Zainul Haque Sikder Women's Medical College Hospital, Dhaka, Bangladesh, between September 2024 and August 2025. According to the selection criteria, 89 adult patients (age $\geq$ 15 years) with hypothyroidism were enrolled in the study. A purposive sampling technique was chosen for this study, which involves a non-random method of selecting participants with specific characteristics. To maintain a focus on the chronic aspects of thyroid disease, patients who had experienced an acute myocardial infarction (MI) or acute renal failure within the last three months were excluded from participation. After the selection of the patients, the aims and procedures of the study were explained to the patients in understandable language including risks and benefits were also made clear to the respondents. Then, written informed consent was obtained from each respondent. The local ethical committee granted ethical approval. Data were collected by using a semi-structured questionnaire through face-to-face interviews. The study population underwent detailed history taking, physical examination, and relevant investigations. Assessment of serum thyroid-stimulating hormone (TSH) and serum lipid profile was done for each respondent.

## Statistical Analysis:

Statistical analysis was conducted using the Windows® software program Statistical Packages for Social Sciences version 25 (SPSS-25) (Chicago, IL, USA). After data collection, all entries were reviewed and cleaned. Quantitative data were reported as percentages, means, and standard deviations, while qualitative data were presented as frequency distributions and percentages. To determine statistical significance, one-way

ANOVA, Kruskal-Wallis test, and Spearman's Correlation Coefficient test were considered according to applicability. A p-value of less than 0.05 was deemed statistically significant.

## Results

A total of 150 subjects were initially included in the study, as determined through interviews and a questionnaire. Out of these, 133 individuals were chosen for the study and grouped based on their thyroid profile and TSH levels. Among these, 44 subjects were euthyroid, whereas 47 and 42 subjects were in the categories of subclinical and clinical hypothyroidism, respectively.

The lipid parameters were compared among euthyroid, subclinical hypothyroid, and clinical hypothyroid individuals. These tables show that all parameters, except HDL, showed statistically significant differences between the euthyroid and hypothyroid groups.

**Table 1: Demographic profile of the study subjects (N=133)**

| | Clinical Hypothyroidism | Subclinical Hypothyroidism | Euthyroid (Control) | p-value |
|---|---|---|---|---|
| n | 42 | 47 | 44 | |
| Age (years) | | | | |
| ≤20 | 1 (2.4) | 4 (8.5) | 8 (18.2) | 0.153 |
| 21 - 30 | 10 (23.8) | 11 (23.4) | 12 (27.3) | |
| 31 - 40 | 18 (42.9) | 14 (29.8) | 11 (25.0) | |
| 41 - 50 | 5 (11.9) | 11 (23.4) | 4 (9.1) | |
| >50 | 8 (19.0) | 7 (14.9) | 9 (20.5) | |
| Mean ± SD | 38.90 ± 10.69 | 37.23 ± 12.96 | 34.95 ± 14.26 | 0.356 |
| Min-max | | | | |
| Gender | | | | |
| Male | 3 (7.1) | 9 (19.1) | 9 (20.5) | 0.175 |
| Female | 39 (92.9) | 38 (80.9) | 35 (79.5) | |
| BMI (kg/m2) | 27.42 ± 4.99 | 25.67 ± 4.49 | 24.66 ± 5.67 | 0.042 |

The ANOVA test was performed on numerical data, while the Chi-Square test was used for categorical data. requency and percentage were used to present categorical data, while numerical data were expressed as mean accompanied by standard deviation**.**

**Table 2: Thyroid hormone profile of the study subjects (N=133)**

| | Clinical Hypothyroidism | Subclinical Hypothyroidism | Euthyroid (Control) | p-value |
|---|---|---|---|---|
| TSH (mIU/mL | 17.15 (54.4) | 7.10 (2.37) | 2.21 (1.28) | <0.001 |
| FT4 (ng/dL) | 0.49 (0.38) | 1.10 (0.30) | 1.20 (0.30) | <0.001 |

The Kruskal-Wallis test was conducted, followed by the Holm-Bonferroni adjustment. Data were reported as the median, accompanied by the interquartile range in parentheses.

| | TSH | FT4 |
|---|---|---|
| A vs B | <0.001 | <0.001 |
| A vs C | <0.001 | <0.001 |
| B vs C | <0.001 | 0.035 |

**Table 3: Lipid profile of the study subjects (N=133)**

| | Clinical Hypothyroidism | Subclinical Hypothyroidism | Euthyroid (Control) | p-value |
|---|---|---|---|---|
| Cholesterol (mg/dL) | 220.98 ± 30.41 | 193.60 ± 39.47 | 174.41 ± 41.11 | <0.001 |
| LDL (mg/dL) | 134.43 ± 28.77 | 114.53 ± 30.83 | 102.09 ± 37.82 | <0.001 |
| HDL (mg/dL) | 36.82 ± 6.94 | 41.83 ± 11.14 | 42.43 ± 8.02 | 0.08 |
| Triglyceride (mg/dL) | 242.21 ± 68.17 | 174.74 ± 85.34 | 147.70 ± 68.63 | <0.001 |

The ANOVA test was conducted, followed by the Bonferroni test. Data were presented as means with standard deviation.

| | Cholesterol | LDL | HDL | Triglyceride |
|---|---|---|---|---|
| A vs B | 0.002 | 0.015 | 0.09 | <0.001 |
| A vs C | <0.001 | <0.001 | 1.000 | <0.001 |
| B vs C | 0.048 | 0.021 | 1.000 | 0.262 |

**Table 4: Correlation of TSH with lipid profiles among patients with hypothyroidism**

| | | r | p-value |
|---|---|---|---|
| TSH | Cholesterol | 0.440 | 0.004 |
| | LDL | 0.287 | 0.006 |
| | HDL | -0.262 | 0.094 |
| | Triglyceride | 0.283 | 0.005 |

The Spearman correlation coefficient test was done.

## Discussion

In this study, the ages of the study subjects ranged from 15 to 70 years. Lipid profiles and BMIs of 44 Euthyroid subjects were compared and evaluated with those of 47 subclinical and 42 clinical hypothyroid subjects. BMI values of euthyroid subjects were either normal or overweight. Out of 89 subjects, clinical hypothyroid subjects had relatively high TSH levels (TSH > 17.15 μIU/ml). Increased BMI (BMI > 25) was observed in individuals in the Clinical and subclinical hypothyroid groups. Subclinical hypothyroid subjects had high TSH levels, i.e.,>7.10μIU/ml. The present study showed a high frequency of increased BMI in subclinical and clinical hypothyroid patients.

In the present study, dyslipidemia was identified in obese subjects, with significantly higher levels of total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TG) compared to non-obese individuals, which aligns with findings from previous studies.[9,12] Lipid abnormalities were prevalent in subclinical and clinical obese hypothyroid subjects.

The present study demonstrated alterations in TC, TGs, and LDL levels in Hypothyroid patients, as shown in Table 3. An increase in BMI was associated with higher TC, TGs, and LDL levels and lower HDL levels, indicating a higher risk of cardiovascular disease in obesity.[13,14] Thyroid hormones are the principal regulators of energy balance. Their role in obesity has been the focus of various scientific studies. The unfavourable effects of high levels of serum TSH on lipid metabolism have been reported, and follow-up studies have shown an increase in the risk of developing atherosclerosis and cardiovascular manifestations in clinical hypothyroid subjects with high normal serum TSH levels.[5,7,15] Genetic and environmental factors play a significant role in the progression of BMI differences, particularly in relation to thyroid function.

According to the American Association of Clinical Endocrinologists (AACE), the number of subclinical subjects with high serum TSH levels is increasing, a finding that is quite significant. TSH > 10 mU/L was found in clinical hypothyroid subjects as per the laboratory reference range. The importance of diagnosing mild thyroid disorders and subclinical hypothyroidism cannot be underestimated because, if not treated, they can severely compromise the quality of life. There is an established association between clinical thyroid dysfunction and weight changes because weight gain is a consistent phenomenon in hypothyroidism. 15. Some studies have concluded that weight gain increases serum levels of TSH, while others showed no relationship between TSH and body weight.[16.] Weight and hypothyroidism. In our study, we found that the percentage of Clinical hypothyroidism patients was higher in terms of obesity compared to subclinical hypothyroidism. This finding aligns with the results of a previous study by Verma et al. (2013), which also showed that obesity was more prevalent (46% vs. 34%) in overt than in subclinical hypothyroidism.[17]

## Limitation:

Limitations of the present study include referral bias and a small sample size.

## Conclusions

Thyroid dysfunction can have important effects on lipid profile in clinical as well as subclinical hypothyroidism. Thyroid hormone profile, lipid profile, and BMI correlate significantly among the hypothyroid subjects. There is a need to monitor patients with hypothyroidism for signs of obesity and various cardiovascular complications. Screening and treatment for subclinical hypothyroidism should be implemented to prevent its adverse effects on lipid metabolism.

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## References

1. Garg Y. A case control study of serum lipid level alterations in subclinical hypothyroid patients. Belgaum, Karnataka: KLE University; 2013.
2. Zimmermann MB, Jooste PL, Pandav CS. Iodine-deficiency disorders. Lancet 2008; 372:1251-62. 3. Yaqoob A. Subclinical hypothyroidism and its consequences. J Public Health Biol Sci 2012; 1:53-60.
4. Kratzsch J, Fiedler GM, Leichtle A, Bru¨gel M, Buchbinder S, Otto L, et al. New reference intervals for thyrotropin and thyroid hormones based on National Academy of Clinical Biochemistry criteria and regular ultrasonography of the thyroid. Clin Chem 2005; 51:1480-6.
5. Duntas LH, Brenta G. The effect of thyroid disorders on lipid levels and metabolism. Med Clin North Am 2012; 96:269-81.
6. Brown MS, Kovanen PT, Goldstein JL. Regulation of plasma cholesterol by lipoprotein receptors. Science 1981; 212: 628-35.
7. Kahaly GJ. Cardiovascular and atherogenic aspects of subclinical hypothyroidism. Thyroid 2000; 10:665-79.
8. Civeira F. Guidelines for the diagnosis and management of heterozygous familial hypercholesterolemia. Atherosclerosis 2004; 173:55-68.
9. Nagila A, Bhatt M, Poudel B, Mahato P, Gurung D, Prajapati S, et al. Thyroid stimulating hormone and its correlation with lipid profile in the obese Nepalese population. J Clin Diagn Res 2008; 2:932-7.
10. Grover GJ, Mellstrom K, Malm J. Therapeutic potential for thyroid hormone receptor-β selective agonists for treating obesity, hyperlipidemia and diabetes. Curr Vascul Pharmacol 2007; 5:141-54.
11. Shaheen S, Zafar K, Khalid A. Prevalence of obesity among teenagers of Rawalpindi/ Islamabad. J Riphah Coll Rehabilitat Sci 2013; 1:23-6.
12. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, Mechanick JI, et al. Clinical practice guidelines for hypothyroidism in adults: co-sponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. Endocrine Pract 2012; 18:988-1028.
13. Mansourian AR. A review on the metabolic disorders of iodine deficiency. Pak J Biol Sci 2011; 14:412-24.

14. Müller O, Krawinkel M. Malnutrition and health in developing countries. Canadian Med Assoc J 2005; 173:279-86

15. Shalitin S, Yackobovitch-Gavan M, Phillip M. Prevalence of thyroid dysfunction in obese children and adolescents before and after weight reduction and its relation to other metabolic parameters. Hormone Res Paediatr 2009; 71:155-61.

16. Sari R, Balci MK, Altunbas H, Karayalcin U. The effect of body weight and weight loss on thyroid volume and function in obese women. Clin Endocrinol 2003; 59:258-62

17. Verma A, Jayaraman M, Kumar HK, Modi KD. Hypothyroidism and obesity? Cause or effect. Saudi Med J. 2008;29:1135–8