



Original Research Article

Biochemical Alterations in Lipid profile and Apolipoproteins in Patient of Hypothyroidism: A cross-sectional study in Western Uttar Pradesh

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Abstract: Hypothyroidism is a common endocrine disorder that significantly influences lipid metabolism and contributes to increased cardiovascular risk. Alterations in conventional lipid parameters and apolipoproteins, particularly apolipoprotein A1 and apolipoprotein B, provide valuable insight into the atherogenic profile associated with thyroid hormone deficiency. **Objectives:** To evaluate biochemical alterations in lipid profile and apolipoproteins among patients with hypothyroidism and to compare these parameters with euthyroid individuals attending a tertiary care hospital in Western Uttar Pradesh. **Methods:** This cross-sectional study was conducted over one year from October 2024 to October 2025 at a tertiary care teaching hospital in District Bijnor, Western Uttar Pradesh. A total of 100 hypothyroid patients and 100 age- and sex-matched euthyroid controls were included. After an overnight fast, blood samples were analyzed for lipid profile parameters and apolipoproteins using standard enzymatic and immunoturbidimetric methods. Data were analyzed using statistical software, and comparisons between groups were made using appropriate statistical tests, with a p-value of less than 0.05 considered statistically significant. **Results:** Hypothyroid patients showed significantly higher levels of total cholesterol, triglycerides, LDL cholesterol, apolipoprotein B, and ApoB/ApoA1 ratio compared to controls. HDL cholesterol and apolipoprotein A1 levels were significantly lower in the hypothyroid group. All observed differences were statistically significant, indicating a pronounced atherogenic lipid and apolipoprotein profile in hypothyroidism. **Conclusion:** Hypothyroidism is associated with significant dyslipidemia and adverse apolipoprotein alterations that may increase cardiovascular risk. Routine assessment of lipid profile along with apolipoproteins in hypothyroid patients is recommended for early identification and prevention of cardiovascular complications.

Keywords: Hypothyroidism; Dyslipidemia; Lipid Profile; Apolipoprotein A1; Apolipoprotein B; ApoB/ApoA1 Ratio; Cardiovascular Risk; Thyroid Hormone Deficiency

INTRODUCTION

Hypothyroidism is one of the most common endocrine disorders worldwide and represents a significant public health concern due to its wide-ranging metabolic effects. Thyroid hormones play a crucial role in regulating basal metabolic rate, lipid metabolism, and cardiovascular homeostasis. Deficiency of thyroid hormones leads to alterations in lipid synthesis, degradation, and transport,

predisposing affected individuals to dyslipidemia and accelerated atherosclerosis. Globally, the prevalence of overt hypothyroidism is estimated to be around 1–2%, while subclinical hypothyroidism affects approximately 4–10% of the adult population, with higher prevalence observed in women and the elderly [1,2].

One of the most consistent biochemical abnormalities observed in hypothyroidism is

dyslipidemia. Reduced thyroid hormone levels lead to decreased expression of hepatic LDL receptors, impaired clearance of low-density lipoprotein cholesterol (LDL-C), and reduced activity of lipoprotein lipase. As a result, patients with hypothyroidism commonly exhibit elevated total cholesterol, LDL-C, triglycerides, and altered high-density lipoprotein cholesterol (HDL-C) levels. These lipid abnormalities significantly increase the risk of coronary artery disease, cerebrovascular disease, and overall cardiovascular morbidity and mortality [3,4].

In addition to conventional lipid parameters, apolipoproteins have emerged as important markers for assessing cardiovascular risk. Apolipoprotein B (ApoB), a structural component of atherogenic lipoproteins, reflects the total number of circulating atherogenic particles, while apolipoprotein A1 (ApoA1), the major protein component of HDL, plays a protective role in reverse cholesterol transport. Several studies suggest that apolipoprotein levels and ApoB/ApoA1 ratio may serve as better predictors of cardiovascular risk than traditional lipid parameters alone, particularly in metabolic and endocrine disorders such as hypothyroidism [5,6].

India bears a substantial burden of thyroid disorders, with hypothyroidism being more prevalent than hyperthyroidism. Population-based studies from different regions of India have reported a prevalence of hypothyroidism ranging from 8% to 11%, which is considerably higher than global estimates. Factors such as iodine intake variability, autoimmune thyroiditis, and lifestyle changes contribute to this increased prevalence. Studies have also documented a high frequency of lipid abnormalities among Indian hypothyroid patients, further amplifying cardiovascular risk in this population [7,8].

Despite growing evidence linking hypothyroidism with dyslipidemia, data on apolipoprotein alterations in hypothyroid patients from North India, particularly Western Uttar Pradesh, remain limited. Regional variations in dietary habits, socioeconomic status, healthcare access, and genetic predisposition may influence lipid and apolipoprotein profiles. Understanding these biochemical alterations is essential for early risk stratification, timely intervention, and prevention of long-term cardiovascular complications.

Therefore, the present cross-sectional study was undertaken to evaluate the biochemical alterations in lipid profile and apolipoproteins among patients with hypothyroidism attending a tertiary care hospital in Western Uttar Pradesh. The findings of this study are expected to provide region-specific data and contribute to improved clinical management and cardiovascular risk assessment in hypothyroid

patients.

MATERIAL AND METHODS

This cross-sectional observational study was conducted over a period of one year from October 2024 to October 2025. The study population consisted of patients attending the Outpatient Department of the Department of Medicine at a tertiary care teaching hospital in District Bijnor, Western Uttar Pradesh. The biochemical investigations were carried out in the Department of Biochemistry, Central Laboratory of Pt. Deendayal Upadhyay District Hospital, a tertiary care government hospital attached to Mahatma Vidur Autonomous State Medical College, Bijnor, Uttar Pradesh.

Patients diagnosed with hypothyroidism based on clinical evaluation and biochemical evidence of thyroid dysfunction were included in the study after obtaining informed consent. Both newly diagnosed and previously known cases of hypothyroidism were considered. Patients with conditions known to affect lipid metabolism such as diabetes mellitus, chronic liver disease, chronic kidney disease, nephrotic syndrome, pregnancy, and those receiving lipid-lowering drugs were excluded from the study to avoid confounding effects. Age- and sex-matched euthyroid individuals attending the same hospital served as the control group.

After an overnight fast of 10–12 hours, venous blood samples were collected under aseptic precautions. Serum was separated by centrifugation and analyzed for thyroid function tests, including serum thyroid-stimulating hormone, free thyroxine, and free triiodothyronine, using standard chemiluminescence immunoassay methods. Based on thyroid function test results, participants were categorized as hypothyroid or euthyroid.

Fasting lipid profile parameters including total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol were estimated using enzymatic colorimetric methods on an automated biochemistry analyzer. Apolipoprotein A1 and apolipoprotein B levels were measured using immunoturbidimetric assay techniques following manufacturer-recommended protocols. All biochemical analyses were performed with appropriate internal quality control measures in place.

The collected clinical and laboratory data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences software. Quantitative variables were expressed as mean with standard deviation, while qualitative variables were expressed as frequency and percentage. Comparison

between hypothyroid patients and euthyroid controls was performed using Student's t-test for continuous variables and Chi-square test for categorical

variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The present study evaluated biochemical alterations in lipid profile and apolipoproteins among hypothyroid patients in comparison with euthyroid controls. A total of 100 hypothyroid patients and 100 age- and sex-matched euthyroid individuals were included. The mean age of hypothyroid patients was comparable to that of controls, with a clear female predominance observed in both groups, reflecting the known higher prevalence of hypothyroidism among women.

Hypothyroid patients demonstrated a markedly deranged lipid profile when compared to euthyroid controls. Mean total cholesterol levels were substantially higher in hypothyroid patients, indicating impaired cholesterol metabolism associated with reduced thyroid hormone activity. Similarly, serum triglyceride levels were significantly elevated among hypothyroid patients, suggesting reduced clearance of triglyceride-rich lipoproteins. Low-density lipoprotein cholesterol levels were also significantly higher in the hypothyroid group, highlighting increased atherogenic potential. In contrast, high-density lipoprotein cholesterol levels were significantly lower in hypothyroid patients, reflecting reduced cardioprotective lipid fractions.

Apolipoprotein analysis further revealed significant alterations in hypothyroid patients. Mean apolipoprotein A1 levels were considerably lower in hypothyroid patients compared to controls, indicating impaired reverse cholesterol transport. Conversely, apolipoprotein B levels were significantly higher in hypothyroid patients, reflecting an increased number of circulating atherogenic lipoprotein particles. The ApoB/ApoA1 ratio, a robust indicator of cardiovascular risk, was markedly elevated in the hypothyroid group, underscoring an unfavorable atherogenic profile.

Statistical analysis confirmed that differences in all lipid parameters and apolipoprotein levels between hypothyroid patients and euthyroid controls were highly significant. The consistent elevation of atherogenic lipids and ApoB, along with reduction in HDL cholesterol and ApoA1, emphasizes the strong association between hypothyroidism and increased cardiovascular risk. These findings reinforce the importance of routine lipid and apolipoprotein assessment in hypothyroid patients for early identification and prevention of cardiovascular complications.

Table 1. Distribution of Study Participants According to Demographic Characteristics

Variable	Hypothyroid patients (n = 100)	Controls (n = 100)
Mean age (years)	41.8 ± 11.6	40.9 ± 10.8
Male	32 (32.0%)	35 (35.0%)
Female	68 (68.0%)	65 (65.0%)
Male : Female ratio	1 : 2.1	1 : 1.9

Table 2. Comparison of Lipid Profile Parameters Between Hypothyroid Patients and Controls

Parameter (mg/dL)	Hypothyroid patients (Mean ± SD)	Controls (Mean ± SD)
Total cholesterol	218.6 ± 42.3	176.4 ± 34.1
Triglycerides	168.9 ± 51.6	122.7 ± 38.4
LDL cholesterol	142.8 ± 36.5	102.3 ± 29.6
HDL cholesterol	41.2 ± 8.4	48.6 ± 9.1

Table 3. Comparison of Apolipoprotein Levels Between Hypothyroid Patients and Controls

Parameter (mg/dL)	Hypothyroid patients (Mean ± SD)	Controls (Mean ± SD)
Apolipoprotein A1	112.4 ± 18.6	136.8 ± 20.3
Apolipoprotein B	124.9 ± 26.8	92.6 ± 21.4
ApoB / ApoA1 ratio	1.12 ± 0.28	0.68 ± 0.19

Table 4. Comparison of Lipid Profile and Apolipoprotein Parameters Between Hypothyroid Patients and Controls with Test of Significance

Parameter	Hypothyroid patients (Mean $\pm$ SD)	Controls (Mean $\pm$ SD)	Mean difference	Test applied	Test value	p-value
Total cholesterol (mg/dL)	218.6 $\pm$ 42.3	176.4 $\pm$ 34.1	42.2	Student's t-test	7.89	<0.001*
Triglycerides (mg/dL)	168.9 $\pm$ 51.6	122.7 $\pm$ 38.4	46.2	Student's t-test	6.41	<0.001*
LDL cholesterol (mg/dL)	142.8 $\pm$ 36.5	102.3 $\pm$ 29.6	40.5	Student's t-test	8.12	<0.001*
HDL cholesterol (mg/dL)	41.2 $\pm$ 8.4	48.6 $\pm$ 9.1	-7.4	Student's t-test	5.36	<0.001*
Apolipoprotein A1 (mg/dL)	112.4 $\pm$ 18.6	136.8 $\pm$ 20.3	-24.4	Student's t-test	8.47	<0.001*
Apolipoprotein B (mg/dL)	124.9 $\pm$ 26.8	92.6 $\pm$ 21.4	32.3	Student's t-test	9.02	<0.001*
ApoB / ApoA1 ratio	1.12 $\pm$ 0.28	0.68 $\pm$ 0.19	0.44	Student's t-test	10.15	<0.001*

* Statistically significant at $p < 0.05$

Figure 1: Percentage Change in Lipid Profile & Apolipoproteins in Hypothyroidism (Vs Controls)

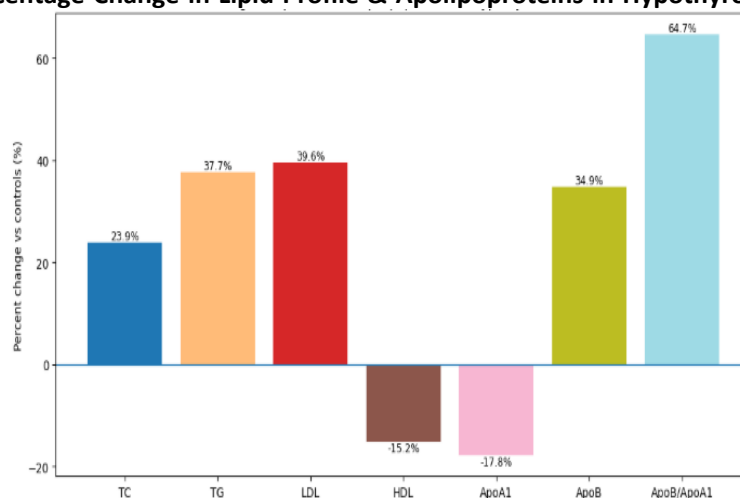
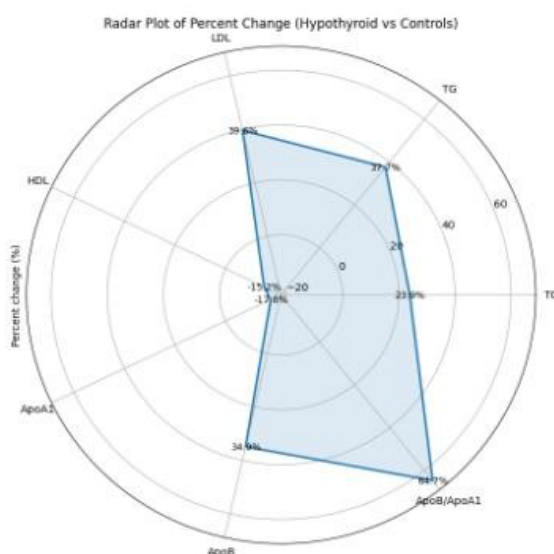


Figure 2: Radar Plot of Percentage Change (Hypothyroid Vs Controls)



DISCUSSION

In this study, hypothyroid patients demonstrated an atherogenic biochemical pattern, with higher mean total cholesterol, LDL-C and triglycerides, along with adverse apolipoprotein changes (higher ApoB and lower ApoA1), supporting the concept that thyroid hormone deficiency shifts lipid transport toward more cholesterol-rich, ApoB-containing particles. This pattern is biologically plausible because hypothyroidism reduces hepatic LDL receptor expression and LDL clearance, increases circulating LDL particle residence time, and may also influence bile acid synthesis and cholesterol efflux, collectively raising total cholesterol and LDL-C levels. Similar directionality has been repeatedly described in major reviews and clinical literature, where overt hypothyroidism is consistently linked to increased TC and LDL-C, with triglycerides variably elevated depending on severity and associated metabolic status. [9,10]

The elevation in triglycerides observed in this study is also consistent with the known effect of hypothyroidism on triglyceride-rich lipoprotein metabolism, where reduced lipoprotein lipase activity and impaired clearance can lead to higher TG levels, particularly in overt disease. A large cross-sectional analysis that compared thyroid function categories reported significantly higher triglycerides in overt hypothyroidism compared with hyperthyroid or euthyroid states, reinforcing that TG elevation can be an important component of hypothyroid dyslipidemia. [11] In parallel, several intervention and observational studies (including population-based datasets) indicate that correction of hypothyroidism with levothyroxine tends to improve lipid parameters, especially total cholesterol and LDL-C, supporting a causal contribution of thyroid status to the dyslipidemic phenotype. [12,13]

Apolipoprotein changes in this study (higher ApoB, lower ApoA1 and an unfavourable ApoB/ApoA1 profile) are clinically meaningful because ApoB reflects the total number of atherogenic lipoprotein particles (VLDL remnants, IDL, LDL), while ApoA1 is a key structural and functional component of HDL. Prior clinical studies evaluating apolipoproteins across thyroid dysfunction states have shown that ApoB tends to be higher in overt and subclinical hypothyroidism compared with hyperthyroid states, while ApoA1 may show less consistent change across categories, depending on sample characteristics and disease severity. [11,14,15] Importantly, expanded lipid profiling studies in hypothyroidism have also demonstrated that “non-conventional” markers (including ApoB and related indices) can rise significantly compared with controls, suggesting that apolipoproteins may capture cardiovascular risk more directly than conventional cholesterol fractions alone in some hypothyroid cohorts. [16]

When comparing this study with regional evidence, cross-sectional work from South Asia has shown a high burden of lipid abnormalities in newly diagnosed hypothyroidism, commonly involving elevated total cholesterol, LDL-C and triglycerides with low HDL-C proportions in many cohorts, aligning with the overall direction of findings here. For example, a tertiary-care descriptive study reported abnormal lipid profiles in a substantial proportion of hypothyroid patients, with frequent elevations of TC, LDL and TG and low HDL patterns, broadly supporting the clinical expectation that hypothyroidism contributes to secondary dyslipidemia in routine practice. [17] Taken together, the present biochemical profile strengthens the clinical implication that hypothyroid patients—even in outpatient settings—represent a group warranting systematic lipid and apolipoprotein evaluation, particularly when additional cardiometabolic risk factors coexist.

CONCLUSION

This cross-sectional study demonstrates that patients with hypothyroidism exhibit significant biochemical alterations in lipid profile and apolipoproteins when compared with euthyroid individuals. Hypothyroid patients showed markedly elevated total cholesterol, triglycerides, LDL cholesterol, apolipoprotein B levels, and ApoB/ApoA1 ratio, along with significantly reduced HDL cholesterol and apolipoprotein A1 levels. These findings indicate a distinctly atherogenic lipid and apolipoprotein pattern associated with hypothyroidism, underscoring an increased risk for cardiovascular morbidity in this population. The results highlight the importance of comprehensive lipid profiling, including apolipoprotein assessment, as part of routine evaluation in patients with hypothyroidism to enable early cardiovascular risk stratification and timely intervention.

Limitations

The study has certain limitations that should be considered while interpreting the results. Being a cross-sectional study, causal relationships between hypothyroidism and lipid or apolipoprotein alterations could not be established. The study was conducted at a single tertiary care center, which may limit the generalizability of the findings to the wider population. The effect of thyroid hormone replacement therapy on lipid and apolipoprotein parameters was not evaluated, and follow-up data were not included. Additionally, dietary patterns, physical activity, and genetic factors influencing lipid metabolism were not assessed, which may have acted as potential confounders.

Recommendations

Based on the findings of this study, routine screening of lipid profile and apolipoproteins is recommended

in all patients diagnosed with hypothyroidism, even in the absence of overt cardiovascular disease. Early identification of dyslipidemia can facilitate timely therapeutic interventions to reduce long-term cardiovascular risk. Incorporation of apolipoprotein measurements, particularly ApoB and ApoB/ApoA1 ratio, may provide better cardiovascular risk assessment than conventional lipid parameters alone. Future studies with larger sample sizes, multicentric design, and longitudinal follow-up are recommended to evaluate the impact of thyroid hormone replacement therapy on lipid and apolipoprotein abnormalities and to establish causal relationships.

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