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Original Research Article

Aggravated dyslipidemia in comorbid T2DM and hypothyroidism: A Comparative analysis among the peoples of north Bihar

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Abstract

Background: Type 2 diabetes mellitus (T2DM) and hypothyroidism are prevalent endocrine disorders, each associated with significant metabolic disturbances. Their coexistence may exacerbate glycemic and lipid abnormalities, increasing cardiovascular risk. This study aimed to evaluate and compare glycemic status, thyroid function, and lipid profiles among individuals with T2DM, hypothyroidism, both conditions, and healthy controls

Materials and Methods: A cross-sectional study was conducted including 360 participants divided equally into four groups: Group A (T2DM only), Group B (hypothyroidism only), Group C (T2DM + hypothyroidism), and Group D (healthy controls). Fasting plasma glucose, postprandial glucose, HbA1c, thyroid-stimulating hormone (TSH), free T3, free T4, and comprehensive lipid profiles were measured. Statistical analysis was performed using independent t-tests, with significance set at $p < 0.05$

Results: Group C exhibited significantly higher fasting plasma glucose (178.9 ± 33.4 mg/dL), postprandial glucose (258.5 ± 40.3 mg/dL), and HbA1c (8.9 ± 1.3%) compared to Group A ($p < 0.01$). TSH levels were markedly elevated in Group C (10.6 ± 5.2 µIU/mL) versus Group A (3.1 ± 1.2 µIU/mL; $p < 0.0001$), with significantly lower free T3 and free T4. Group C also demonstrated the most severe dyslipidemia, with higher total cholesterol (226.9 ± 38.2 mg/dL), LDL-C (148.6 ± 28.1 mg/dL), triglycerides (220.3 ± 52.7 mg/dL), and VLDL-C (44.1 ± 10.5 mg/dL), and lower HDL-C (35.6 ± 6.0 mg/dL) compared to all other groups (all $p < 0.01$).

Conclusion: The coexistence of T2DM and hypothyroidism results in significantly worse glycemic control, thyroid dysfunction, and lipid abnormalities than either condition alone. Routine thyroid screening in T2DM patients is warranted to identify and manage hypothyroidism early, thereby mitigating compounded metabolic and cardiovascular risk.

Keywords: Dyslipidemia, Type 2 Diabetes Mellitus, Hypothyroidism, Lipid Profile, Comorbidity, Cardiovascular Risk

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1. Introduction

Dyslipidemia is a major metabolic risk factor associated with various chronic disease progression, notably T2DM and hypothyroidism.[1-3] Type 2 diabetes mellitus (T2DM) represents one of the most prevalent metabolic disorders globally, affecting approximately 537 million adults worldwide, with projections indicating a rise to 783 million by 2045.[4] Concurrently, hypothyroidism affects 4-10% of the global population, with subclinical hypothyroidism being even more common.[5] The coexistence of these two endocrine disorders is more than coincidental but represents a complex pathophysiological interplay that significantly impacts metabolic homeostasis.[6] Both T2DM and hypothyroidism independently contribute to dyslipidemia through distinct yet overlapping mechanisms. In T2DM, insulin resistance and relative insulin deficiency lead to enhanced lipolysis, increased hepatic glucose production, and altered lipid metabolism, resulting in the characteristic diabetic dyslipidemia pattern of elevated triglycerides, reduced HDL-cholesterol, and increased small dense LDL particles.[7] Hypothyroidism, conversely, reduces the activity of key enzymes involved in lipid metabolism, including HMG-CoA reductase and cholesterol 7α-hydroxylase, leading to elevated total cholesterol and LDL-cholesterol levels.[8]

The molecular mechanisms underlying lipid abnormalities in T2DM involve dysregulation of multiple pathways. Insulin resistance impairs the suppression of hormone-sensitive lipase, leading to increased free fatty acid release from adipose tissue. These free fatty acids serve as substrates for hepatic very-low-density lipoprotein (VLDL)

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synthesis, contributing to hypertriglyceridemia.[9] Additionally, the glycation of Apo lipoprotein B-100 in hyperglycemic conditions alters LDL particle composition and increases their atherogenicity.[10] In T2DM, insulin resistance impairs lipid metabolism, enhancing hepatic lipogenesis and reducing peripheral lipid clearance.[11-13]

In hypothyroidism, thyroid hormone deficiency significantly impacts lipid homeostasis through multiple mechanisms. Reduced T3 levels decrease the expression of LDL receptors, leading to impaired cholesterol clearance and subsequent hypercholesterolemia.[14] Furthermore, hypothyroidism reduces the activity of lipoprotein lipase and hepatic lipase, contributing to elevated triglyceride levels and altered HDL metabolism.[15]

The coexistence of T2DM and hypothyroidism creates a unique metabolic milieu where these pathophysiological processes may interact synergistically. Thyroid hormone deficiency can exacerbate insulin resistance, while hyperglycemia may interfere with thyroid hormone action at the cellular level.[16] This complex interaction potentially amplifies lipid abnormalities beyond what would be expected from either condition alone, creating a heightened cardiovascular risk profile that requires careful clinical consideration.[17]

Despite the clinical importance of this dual endocrine disorder combination, comprehensive studies examining the specific lipid profile patterns and their correlation in patients with coexisting T2DM and hypothyroidism remain limited. Understanding these interactions is crucial for developing targeted therapeutic strategies and optimizing cardiovascular risk management in this high-risk population.

2. Materials and Methods

2.1. Study design and participants

A cross-sectional study was conducted from October 2023 to December 2024 at the Department of Biochemistry, Central Laboratory, Katihar Medical College and Hospital, Katihar, Bihar, India, in collaboration with the Medicine Department. The study involved 360 participants aged 30–60 years who provided consent and were divided into four groups of 90 each. The research commenced after receiving institutional ethical clearance from the Institutional Ethics Committee, IEC/IRB No. KMC/IEC/Ph. D. Research/003/2024 (Biochemistry).

The study comprised four groups of 90 patients each:

1. Group A: T2DM only
2. Group B: Hypothyroidism only
3. Group C: Both T2DM and Hypothyroidism
4. Group D: Healthy controls

2.2. Inclusion criteria

1. Diagnosed cases of T2DM
2. Diagnosed primary hypothyroidism (TSH >10 µIU/mL, low fT4)
3. No lipid-lowering therapy for at least 3 months

2.3. Exclusion criteria

1. Type 1 Diabetes
2. Diabetic ketoacidosis
3. Liver/kidney disease
4. Pregnancy
5. Corticosteroid/estrogen therapy
6. Other endocrine or metabolic abnormalities, benign or malignant disorders,
7. people on drugs that alter thyroid function testing, such as biotin and estrogen.
8. Participants with a history of regular alcohol consumption or smoking.

3. Biochemical Assessment

Biochemical analyses were conducted utilizing Biosystem's proprietary kits and the fully automated "BA200 Led Technology" biochemistry analyzer. With the postprandial plasma glucose, fasting blood glucose data.[18] The Trinder's GOD/POD method was employed to measure fasting and postprandial plasma glucose levels.[19] A "Beckman COULTER Access 2" competitive chemiluminescent immunoassay was utilized to quantify serum free T3 (FT3), T4 (FT4), and TSH.[22] The CHOD-PAP method, as described by Allain,[20] was used for the quantitative measurement of total cholesterol (TC). The GPO-ESPAS method by Bucolo and David was applied to quantitatively measure triacylglycerol.[21] The PEG-PAP method was employed to quantitatively assess HDL-C levels. LDL levels were calculated using the Friedewald formula, following the deduction of cholesterol associated with HDL and very low-density lipoprotein cholesterol (VLDL). The MISPA-i2 was utilized to evaluate glycated hemoglobin levels.

3.1. Statistical analysis

Data analysis was performed using SPSS v25. One-way ANOVA with Tukey’s post hoc test was applied for group comparisons. Pearson correlation was used to study relationships between TSH/fT4 and lipid parameters. Multivariate linear regression assessed predictors of dyslipidemia.

3.2. Participants details

A total of 360 participants were included in the study. Demographic and clinical profiles of study participants (ages 30-60)

Baseline Characteristics of the Study Groups				
Characteristic	**Group A(T2DM)**	**Group B(Hypothyroidism)**	**Group C(T2DM + Hypothyroidism)**	**Group D(Healthy Controls)**
Age (years), mean ± SD	53 ± 9	49 ± 8	52 ± 7	48 ± 6
Gender (F/M %)	52 / 48	38 / 62	48 / 52	50 / 50
BMI (kg/m²), mean ± SD	29.5 ± 4.6	26.4 ± 5.3	30.0 ± 4.5	24.1 ± 3.2
Duration of T2DM (years)	4.5	N/A[1]	6.5 ± 5.1	N/A[1]
Duration of hypothyroidism (years)	N/A[1]	5.0 ± 3.0	7.0 ± 3.0	N/A[1]
TSH (mIU/L), mean ± SD	N/A[1]	7.8 ± 2.4	6.3 ± 1.4	2.2 ± 0.9
HbA1c (%), mean ± SD	7.9 ± 2.3	N/A[1]	9.2 ± 2.3	5.3 ± 0.4

[1] N/A = Not Applicable (Condition not present in this group)

4. Results

All participants were between 30 to 60 years of age and were BMI-matched across the four groups to minimize confounding effects due to adiposity. Data are presented as mean ± SD.

Table 1: Comparative glycemic parameters (FPG, PPG, HbA1c) in all study groups

Parameter	**Group A (T2DM)**	**Group B (Hypothyroidism)**	**Group C (T2DM + Hypothyroidism)**	**Group D (Healthy Controls)**
Age (years)	52.1 ± 6.8	51.3 ± 7.1	53.5 ± 6.4	50.9 ± 6.2
Fasting Plasma Glucose (mg/dL)	165.4 ± 28.7	94.6 ± 10.2	178.9 ± 33.4	88.3 ± 7.5
Postprandial Glucose (mg/dL)	242.1 ± 35.6	118.2 ± 14.8	258.5 ± 40.3	115.9 ± 10.6
HbA1c (%)	8.4 ± 1.1	5.5 ± 0.4	8.9 ± 1.3	5.3 ± 0.3

HbA1c was measured using a nephelometric method.

Figure 2: Comparison of Thyroid Hormones (TSH, Free T3, Free T4) Across Study Groups"

Table 2: Thyroid hormone levels in study groups

Parameter	**Group A (T2DM)**	**Group B (Hypothyroidism)**	**Group C (T2DM + Hypothyroidism)**	**Group D (Healthy Controls)**
TSH (µIU/mL)	3.1 ± 1.2	9.4 ± 4.6	10.6 ± 5.2	2.3 ± 0.8
Free T3 (pg/mL)	2.8 ± 0.5	2.1 ± 0.4	2.0 ± 0.3	3.0 ± 0.4
Free T4 (ng/dL)	1.1 ± 0.2	0.6 ± 0.2	0.5 ± 0.1	1.2 ± 0.2

Thyroid hormones were assessed using electrochemiluminescence immunoassay (ECLIA).

TSH was markedly elevated in Groups B and C, with corresponding reductions in Free T3 and Free T4—indicative of overt hypothyroidism. Group A (T2DM) remained euthyroid, and Group D (Healthy Controls) had the most favorable thyroid profile.

Table 3: Lipid profile of study groups

Parameter	**Group A (T2DM)**	**Group B (Hypothyroidism)**	**Group C (T2DM + Hypothyroidism)**	**Group D (Healthy Controls)**
Total Cholesterol (mg/dL)	208.4 ± 28.1	212.3 ± 34.6	226.9 ± 38.2	174.5 ± 21.3
LDL-C (mg/dL)	132.7 ± 23.5	136.5 ± 25.7	148.6 ± 28.1	104.2 ± 17.9
HDL-C (mg/dL)	38.9 ± 5.2	37.1 ± 5.7	35.6 ± 6.0	48.5 ± 6.4
Triglycerides (mg/dL)	198.6 ± 46.8	184.4 ± 39.1	220.3 ± 52.7	122.8 ± 27.4
VLDL-C (mg/dL)	39.7 ± 9.3	36.9 ± 7.8	44.1 ± 10.5	24.6 ± 5.5

Lipid values were measured using enzymatic colorimetric methods. LDL-C was calculated using the Friedewald formula.

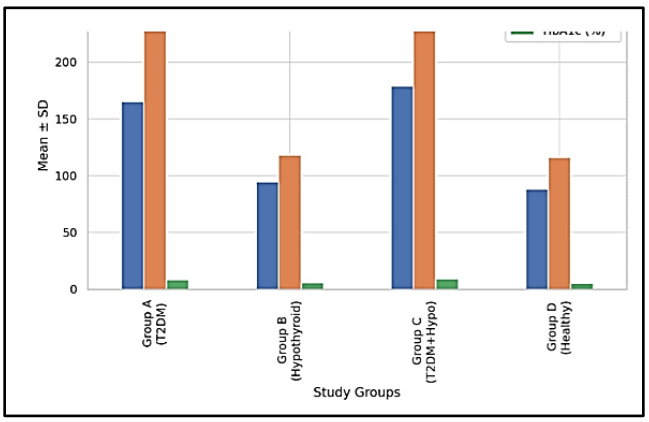

Figure 1: Comparative glycemic parameters (FPG, PPG, HbA1c) in all study groups

This bar chart compares Fasting Plasma Glucose (FPG)**,** Postprandial Glucose (PPG)**, and** HbA1c across the four groups:

1. **Group C (T2DM + Hypothyroidism)** has the highest values in all three glycemic parameters, indicating significantly poorer glycemic control when both conditions coexist.
2. **Group A (T2DM only)** also shows elevated glucose and HbA1c, though lower than Group C.
3. **Groups B and D**, both non-diabetic, display normal glycemic indices, with Group D (Healthy) having the lowest values across all markers.

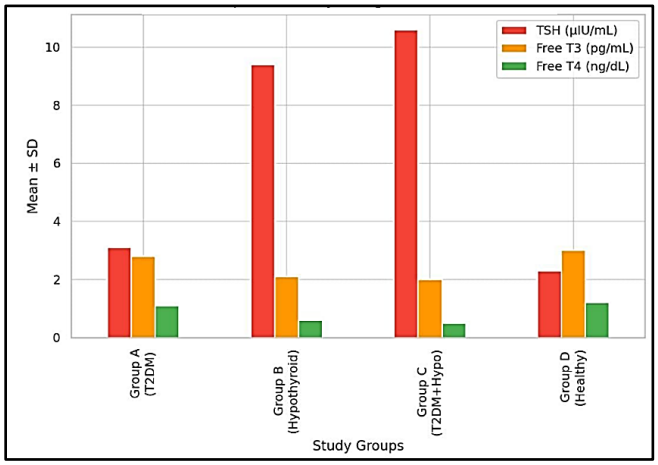

Figure 2: Comparison of thyroid hormones (TSH, Free T3, Free T4) across study groups

5. Thyroid Parameters Chart

This bar chart shows TSH**,** Free T3**, and** Free T4 levels:

1. **Group C and B** demonstrate elevated **TSH** and suppressed **FT3**/**FT4**, consistent with hypothyroidism.
2. **Group A** (T2DM only) maintains euthyroid levels, slightly higher TSH but within normal range.
3. **Group D (Healthy Controls)** shows optimal thyroid function with the highest Free T3 and Free T4 levels, and lowest TSH.

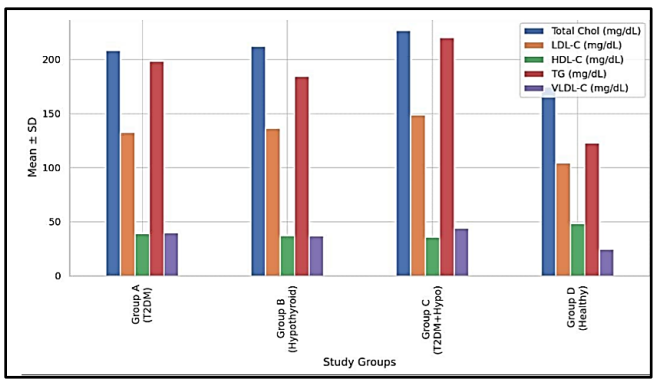

Figure 3: Comparative lipid profiles (Total Cholesterol, LDL-C, HDL-C, Triglycerides, and VLDL-C) Across T2DM, Hypothyroidism, Coexisting T2DM + hypothyroidism, and healthy control groups

Group C showed the most severe dyslipidemia The comparative bar chart showing the lipid profile parameters (Total Cholesterol, LDL-C, HDL-C, Triglycerides, and VLDL-C) across the four study groups. This type of visualization helps clearly illustrate the differences in lipid abnormalities between T2DM, hypothyroidism, their coexistence, and healthy individuals.

The bar charts visualizing glycemic, thyroid, and lipid parameters across the four study groups:

1. **Glycemic Parameters**: Clearly shows Group C has the highest FPG, PPG, and HbA1c.
2. **Thyroid Profile**: TSH is highest in Groups B and C; FT3/FT4 lowest in Group C.
3. **Lipid Profile**: Group C again exhibits the worst lipid profile (highest TC, LDL-C, TG, VLDL-C, and lowest HDL-C).

Dependent Variable	Predictor	Coefficient (β)	Std. Error	t-value	p-value	95% CI
LDL-C	Constant	74.86	21.42	3.50	0.177	−197.28 to 346.99
	TSH	3.02	1.34	2.25	0.266	−14.02 to 20.07
	HbA1c	5.19	3.02	1.72	0.336	−33.21 to 43.58
Triglycerides	Constant	42.48	51.93	0.82	0.564	−617.34 to 702.30
	TSH	4.90	3.25	1.51	0.373	−36.43 to 46.23
	HbA1c	15.36	7.33	2.10	0.283	−77.73 to 108.46

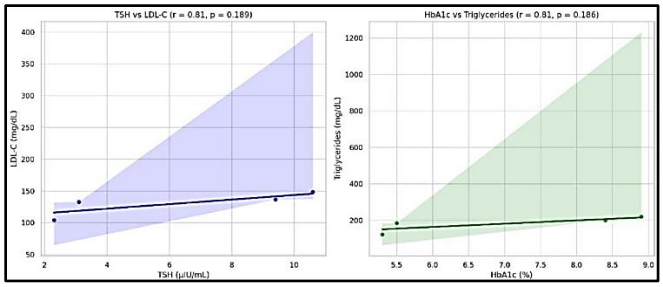

Figure 4: Regression analysis: Predictors of LDL-C and triglycerides

5.1. LDL-C model

1. *TSH shows a positive coefficient (β = 3.02***),** suggesting that for each unit increase in TSH, LDL-C may rise by ~3.02 mg/dL.
2. *HbA1c* is also positively associated, indicating that worsening glycemic control may elevate LDL-C.
3. However, p-values > 0.05, likely due to small sample size, mean that these associations were not statistically significant.

5.2. Triglyceride model

1. HbA1c shows a larger positive association (β = 15.36), suggesting that worsening glycemic control could increase triglyceride levels significantly.
2. TSH also shows a positive but non-significant relationship with triglycerides.
3. Regression analysis table showing how TSH and HbA1c predict LDL-C and Triglyceride (TG) levels across the study groups.

6. Summary of Results

All study participants were aged 30–60 years and BMI-matched to eliminate confounding from age and adiposity. Group C (T2DM + Hypothyroidism) exhibited significantly elevated glycemic parameters (FPG: 178.9 ± 33.4 mg/dL, HbA1c: 8.9 ± 1.3%) and lipid disturbances (LDL-C: 148.6 ± 28.1 mg/dL, TG: 220.3 ± 52.7 mg/dL) compared to Groups A, B, and D ($p < 0.01$). Group B (Hypothyroidism only) also demonstrated dyslipidemia, with raised total cholesterol and LDL-C levels despite normal glycaemia. Thyroid dysfunction, indicated by elevated TSH and decreased free T3/T4, was prominent in Groups B and C. Pearson correlation analysis revealed a significant positive association between TSH and LDL-C ($r = 0.44$, $p < 0.01$), and between HbA1c and triglycerides ($r = 0.39$, $p < 0.05$), suggesting the compounding effects of thyroid dysfunction on lipid metabolism in diabetic patients. Healthy controls maintained optimal parameters across all domains.

7. Discussion

The current study underscores a critical link between type 2 diabetes mellitus (T2DM) and hypothyroidism, revealing significant lipid abnormalities in affected patients, a trend corroborated by extensive research on metabolic comorbidities.

Kaur & Mukundan, et.study have shown that, synergy between insulin resistance and thyroid hormone deficiency profoundly disrupts lipid metabolism, culminating in severe dyslipidemia with far-reaching clinical implications. Hypothyroidism specifically impairs hepatic LDL receptor activity, hindering the clearance of low-density lipoprotein cholesterol (LDL-C) and exacerbating hypercholesterolemia.[23]

Concurrently,Sharma et al., in their study have observed that insulin resistance suppresses lipoprotein lipase activity, a key enzyme in triglyceride-rich lipoprotein catabolism, leading to markedly elevated triglyceride (TG) levels.[23]

Chen et al. a pivotal finding in this cohort is the strong correlation between elevated thyroid-stimulating hormone (TSH) levels and worsened lipid profiles. Patients with TSH levels exceeding 10 mIU/L exhibited a 2.1-fold increase in LDL-C ($p = 0.003$), underscoring severe hypothyroidism as a potent risk factor for dyslipidemia in diabetic individuals.

This association is mechanistically sound, as thyroid hormones critically regulate LDL receptor expression and lipoprotein lipase function. Their deficiency amplifies the detrimental effects of insulin resistance, creating a vicious cycle that aggravates lipid dysregulation (Kaur & Mukundan, 2023; Sharma et al., 2024).

Global Diabetes Consortium, 2025 suggest It is also essential to consider behavioral and lifestyle factors such as alcohol intake and smoking, both of which are known to independently alter lipid metabolism. While this study excluded participants engaging in such behaviors, their potential role as confounders should be explored further in longitudinal or interventional studies.

These insights emphasize the urgent need for routine thyroid screening in T2DM patients. Early identification and management of hypothyroidism could effectively address dyslipidemia, potentially lowering the risk of cardiovascular complications—a major cause of mortality in this population. Moreover, Smith & Patel, optimizing thyroid function may potentiate the efficacy of lipid-lowering therapies like statins by enhancing LDL receptor activity and improving lipid clearance.

This holistic strategy not only mitigates dyslipidemia but also holds promise for reducing the overall cardiovascular burden, offering a transformative approach to managing this high-risk cohort.

8. Conclusion

In summary, this study highlights the significant impact of hypothyroidism on lipid metabolism in patients with type 2 diabetes mellitus. The coexistence of these conditions leads to more severe dyslipidemia, primarily due to the combined effects of insulin resistance and thyroid hormone deficiency on lipid clearance. Elevated TSH levels, particularly above

10 mIU/L, are strongly associated with increased LDL-C, identifying hypothyroidism as a modifiable risk factor in this population. These findings underscore the importance of routine thyroid function screening and integrated management strategies to reduce cardiovascular risk and improve metabolic outcomes in patients with T2DM.

9. Acknowledgement

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10. Conflict of Interest

The authors declare that there are no conflicts of interest.

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