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RESEARCH ARTICLE

Thyroid dysfunction and metabolic dysregulation: A cross-sectional study of hormonal and glycemic parameters

Pronoy Kumer Sarker[1], Nisat Sultana[2], Hafizul Islam[3], Md Samiul Bashir[4], Md. Sujon Ali[5*]

[1] Pronoy Kumer Sarker, Department of Laboratory Medicine, Popular Diagnostic Centre Limited, Dhaka, Bangladesh

[2] Nisat Sultana, Department of Microbiology, Stemz Health Care Limited, Dhaka, Bangladesh

[3] Hafizul Islam, Department of Transfusion Services, The Ohio State University- Wexner Medical Center, USA.

[4] Md Samiul Bashir, Department of Laboratory Medicine, Institute of Health Technology, Kurigram, Bangladesh

[5] Md. Sujon Ali, Department of Medical Biotechnology, University of Technology Sydney (UTS), Sydney, Australia*

***Corresponding author:** Md. Sujon Ali, msujonali07@gmail.com

ABSTRACT

Background: Thyroid dysfunction, encompassing hypothyroidism and hyperthyroidism, has been implicated in metabolic dysregulation, including disturbances in glucose and lipid metabolism. Given the rising global prevalence of metabolic disorders such as diabetes and dyslipidemia, understanding the interplay between thyroid hormones and metabolic health is crucial. This study aimed to investigate the association between thyroid dysfunction and metabolic parameters in a clinical population.

Methods: A cross-sectional study was conducted at the Diabetic Care Center, Dhaka, from January to June 2023, involving 140 adult participants. Thyroid function (TSH, FT_3, FT_4), glycemic markers (FBG, ABF, HbA1c), and lipid profiles were assessed. Statistical analyses included correlation tests and multivariate regression to evaluate relationships between thyroid and metabolic parameters.

Results: The study revealed that hypothyroid participants had significantly higher fasting blood glucose (124.7 ± 18.9 mg/dL vs. 108.3 ± 14.5 mg/dL, $p < 0.01$), postprandial glucose (165.5 ± 25.4 mg/dL vs. 143.9 ± 20.2 mg/dL, $p < 0.01$), and HbA1c (7.4 ± 1.3% vs. 6.5 ± 1.1%, $p < 0.01$) compared to euthyroid individuals. Additionally, hypothyroidism was associated with elevated total cholesterol (218.6 ± 34.5 mg/dL vs. 190.4 ± 28.6 mg/dL, $p < 0.05$) and LDL (141.7 ± 27.8 mg/dL vs. 119.5 ± 23.3 mg/dL, $p < 0.05$), alongside reduced HDL (39.2 ± 7.3 mg/dL vs. 44.7 ± 8.9 mg/dL, $p = 0.04$). Hyperthyroid patients also exhibited metabolic disturbances, including higher FBG (116.1 ± 16.8 mg/dL) and triglycerides (176.5 ± 38.7 mg/dL). Correlation analysis demonstrated a significant association between TSH and HbA1c ($r = 0.38$, $p < 0.01$), reinforcing the role of thyroid dysfunction in metabolic dysregulation.

Conclusion: Thyroid dysfunction, particularly hypothyroidism, is significantly associated with impaired glycemic control and dyslipidemia. Routine thyroid screening in metabolic disorder patients and integrated management strategies are recommended to mitigate cardiovascular and diabetic risks.

Keywords: Thyroid dysfunction; metabolic dysregulation; hypothyroidism; glycemic control; dyslipidemia

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1. Introduction

The thyroid gland is a vital endocrine organ responsible for the synthesis and release of thyroid hormones—primarily thyroxine (T_4) and triiodothyronine (T_3)—under the regulation of thyroid-stimulating hormone (TSH) produced by the anterior pituitary[1]. These hormones are integral to maintaining basal metabolic rate and modulating a wide array of physiological functions including thermogenesis, protein synthesis, lipid metabolism, cardiovascular activity, and glucose homeostasis[2,3]. Given its widespread systemic influence, even subtle alterations in thyroid hormone levels can produce significant metabolic consequences.

Thyroid dysfunction encompasses a spectrum of disorders, most commonly hypothyroidism (deficient thyroid hormone production) and hyperthyroidism (excessive thyroid hormone production), as well as subclinical forms where laboratory abnormalities exist without overt clinical symptoms[4]. According to global estimates, thyroid disorders affect hundreds of millions of individuals, with hypothyroidism being particularly prevalent in iodine-deficient regions and autoimmune thyroiditis contributing significantly to disease burden worldwide. Despite often being underdiagnosed or misattributed to nonspecific symptoms such as fatigue and weight changes, thyroid dysfunction can substantially impact metabolic processes[5].

On the other hand, metabolic dysregulation refers to disturbances in normal metabolic processes, most notably those related to glucose and lipid metabolism. It includes conditions such as insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus (T2DM), dyslipidemia, and metabolic syndrome[6]. These disorders represent a growing global health crisis, driven by sedentary lifestyles, poor dietary habits, rising obesity rates, and increased life expectancy. The endocrine system, particularly the interplay between thyroid, pancreatic, and adrenal hormones, is central to maintaining metabolic balance, and disturbances in one domain can have cascading effects across others[7].

Mounting evidence highlights a bidirectional relationship between thyroid function and metabolic health. For instance, hypothyroidism has been linked with reduced insulin sensitivity, hyperlipidemia, elevated body mass index (BMI), and central obesity—all of which are components of metabolic syndrome[8]. Conversely, hyperthyroidism may lead to increased hepatic glucose output, enhanced intestinal glucose absorption, reduced insulin half-life[9], and muscle wasting, potentially aggravating glycemic control in predisposed individuals. The relationship is further complicated by shared pathophysiological pathways, such as chronic low-grade inflammation, oxidative stress, and autoimmune mechanisms[10].

Moreover, thyroid hormones exert direct effects on pancreatic β-cell function[11], hepatic glucose production, adipocyte metabolism, and peripheral glucose uptake via modulation of insulin receptor expression and activity[12-14]. In individuals with diabetes, abnormal thyroid function can impair glycemic control, exacerbate complications such as neuropathy and dyslipidemia, and hinder the effectiveness of pharmacological therapies[15]. Additionally, undiagnosed thyroid disorders may mimic or obscure metabolic symptoms, making accurate diagnosis and timely intervention essential. The present cross-sectional study aims to investigate the relationship between thyroid dysfunction and metabolic dysregulation by analyzing key hormonal (TSH, FT_3, FT_4) and glycemic (FBG, HbA1c, insulin) parameters in a defined population.

2. Methodology

2.1. Study design and setting

This cross-sectional observational study was conducted at the Diabetic Care Center, Dhaka, over a six-month period from January 2023 to June 2023. The center primarily serves patients with endocrine and

metabolic disorders, making it an appropriate setting to explore the relationship between thyroid dysfunction and metabolic dysregulation. The study was designed to evaluate correlations between hormonal imbalances and glycemic parameters in individuals presenting for routine check-ups, follow-up assessments, or newly diagnosed endocrine/metabolic issues.

2.2. Study population and sample size

A total of 140 adult patients aged 18 years and above were included in the study using a convenience sampling method. Participants were eligible if they were undergoing assessment for thyroid function and/or glycemic status and had provided written informed consent.

2.3. Inclusion and exclusion criteria

Inclusion Criteria:

- Adults aged 18 years and above
- Patients undergoing evaluation for thyroid and/or glycemic markers
- Individuals who provided informed consent

Exclusion Criteria:

- Pregnant women
- Individuals with chronic liver or kidney disease or malignancy
- Patients on corticosteroids, immunosuppressive therapy, or hormone therapy (excluding thyroid hormones)
- Patients with recent acute illness or hospitalization in the last three months

2.4. Blood sample collection and laboratory analysis

Fasting venous blood samples were collected from all participants following an overnight fast of 8 to 12 hours. Laboratory analysis was carried out at the central biochemistry laboratory of the Diabetic Care Center, adhering to standardized quality control protocols[16].

The biochemical parameters measured included:

- Fasting Blood Glucose (FBG) and Postprandial Blood Glucose (ABF) using the glucose oxidase-peroxidase method
- HbA1c using high-performance liquid chromatography (HPLC)
- Thyroid function tests (TSH, FT_3, FT_4) using chemiluminescent immunoassay (CLIA)
- Lipid profile including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides using enzymatic colorimetric methods

All collected samples were processed following biosafety standards, and results were recorded confidentially.

2.5. Study variables

- Independent Variables: Age, sex, BMI, blood pressure, clinical symptoms, and current medications
- Dependent Variables:

Hormonal Parameters: TSH, FT_3, FT_4

Glycemic Parameters: FBG, ABF, HbA1c

Lipid Parameters: Total cholesterol, LDL, HDL, triglycerides

2.6. Statistical analysis

Data entry and organization were performed using Microsoft Excel. Statistical analysis was conducted using SPSS software (version [Insert Version, e.g., 25.0]). Descriptive statistics, such as means, standard deviations, frequencies, and percentages, were used to summarize both continuous and categorical variables.

Comparative analyses were conducted to examine differences between thyroid status groups and metabolic markers, using appropriate statistical tests based on data distribution. Correlation analyses were used to explore relationships between thyroid hormone levels and glycemic indices. Multivariate analysis and regression models were used to identify independent predictors of metabolic dysregulation while adjusting for confounding variables such as age, sex, and BMI. A p-value of <0.05 was considered statistically significant in all statistical tests performed.

2.7. Ethical considerations

The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment, ensuring voluntary participation and confidentiality of personal data.

3. Results

A total of 140 participants were enrolled, of which 82 (58.6%) were female and 58 (41.4%) were male. The mean age of the participants was 46.7 ± 12.4 years (range: 18–75 years). The mean BMI was 27.8 ± 4.6 kg/m², indicating that the majority of participants were either overweight or obese. Hypertension was observed in 62 (44.3%) participants. Clinical symptoms suggestive of thyroid or metabolic disturbances, such as fatigue, weight fluctuations, or palpitations, were reported by 89 participants (63.6%).

Table 1. Demographic and clinical characteristics of the study population (n = 140)

Parameter	Mean ± SD / n (%)
Age (years)	46.7 ± 12.4
• 18–39 years	42 (30.0%)
• 40–59 years)	71 (50.7%)
• ≥60 years)	27 (19.3%)
Gender	
• Female	82 (58.6%)
• Male	58 (41.4%)
BMI (kg/m²)	27.8 ± 4.6
Hypertension	62 (44.3%)
Symptomatic Patients	89 (63.6%)
Asymptomatic Patients	51 (36.4%)

Table 2 presents the distribution of participants based on thyroid function status. The majority of the study population (61.4%) were euthyroid, followed by 27.1% who had hypothyroidism (including subclinical forms), and 11.4% with hyperthyroidism. The mean TSH level was significantly elevated in the hypothyroid group (7.4 ± 2.3 mIU/L), indicating impaired thyroid hormone feedback. In contrast, the hyperthyroid group

had a suppressed mean TSH value of 0.15 ± 0.08 mIU/L, consistent with negative feedback due to elevated thyroid hormones. Additionally, FT_3 and FT_4 values aligned with the expected patterns: lower in hypothyroidism and higher in hyperthyroidism. These findings confirm accurate biochemical classification and support the hormonal basis for metabolic comparison in the study.

Table 2. Distribution of thyroid function status (n = 140)

Thyroid Status	n (%)	Mean TSH (mIU/L)	Mean FT_3 (pg/mL)	Mean FT_4 (pmol/L)
Euthyroid	86 (61.4%)	2.1 ± 0.9	3.5 ± 0.8	15.2 ± 3.7
Hypothyroid	38 (27.1%)	7.4 ± 2.3	2.8 ± 0.9	13.1 ± 2.8
Hyperthyroid	16 (11.4%)	0.15 ± 0.08	4.7 ± 1.1	22.3 ± 4.1

Table 3 illustrates the relationship between glycemic parameters (FBG, ABF, and HbA1c) and thyroid function. Participants with hypothyroidism exhibited significantly higher fasting blood glucose (124.7 ± 18.9 mg/dL), postprandial glucose (165.5 ± 25.4 mg/dL), and HbA1c (7.4 ± 1.3%) compared to euthyroid individuals ($p < 0.01$ for all). These results suggest that hypothyroidism may contribute to impaired glucose metabolism, possibly through increased insulin resistance and reduced glucose clearance. Interestingly, although hyperthyroid patients had lower HbA1c values (6.3 ± 0.9%), their FBG levels remained elevated, indicating a complex interplay of increased hepatic glucose output and altered insulin dynamics in hyperthyroidism.

Table 3. Glycemic parameters by thyroid function status (n = 140)

Parameter	Euthyroid (n = 86)	Hypothyroid (n = 38)	Hyperthyroid (n = 16)	p-value
FBG (mg/dL)	108.3 ± 14.5	124.7 ± 18.9	116.1 ± 16.8	<0.01
ABF (mg/dL)	143.9 ± 20.2	165.5 ± 25.4	157.6 ± 19.3	<0.01
HbA1c (%)	6.5 ± 1.1	7.4 ± 1.3	6.3 ± 0.9	<0.01

Table 4 compares lipid parameters among the three thyroid groups. Hypothyroid patients had the most unfavorable lipid profiles, with significantly higher total cholesterol (218.6 ± 34.5 mg/dL) and LDL cholesterol (141.7 ± 27.8 mg/dL) compared to euthyroid participants. HDL cholesterol was notably lower in the hypothyroid group (39.2 ± 7.3 mg/dL), while triglyceride levels were elevated in both hypothyroid and hyperthyroid groups. These results underscore the atherogenic potential of thyroid dysfunction, especially hypothyroidism, which is known to reduce lipid clearance and alter lipoprotein metabolism. The dyslipidemia observed in hyperthyroidism, although milder, may be due to enhanced lipid turnover and hepatic stimulation.

Table 4. Lipid profile by thyroid function status (n = 140)

Lipid Parameter	Euthyroid (n = 86)	Hypothyroid (n = 38)	Hyperthyroid (n = 16)	p-value
Total Cholesterol (mg/dL)	190.4 ± 28.6	218.6 ± 34.5	196.2 ± 30.1	<0.05
LDL (mg/dL)	119.5 ± 23.3	141.7 ± 27.8	123.9 ± 22.5	<0.05
HDL (mg/dL)	44.7 ± 8.9	39.2 ± 7.3	42.5 ± 9.0	0.04
Triglycerides (mg/dL)	158.6 ± 35.3	182.3 ± 41.2	176.5 ± 38.7	0.03

Table 5 presents the correlation coefficients between thyroid hormone levels and key metabolic indicators. A moderate positive correlation was found between TSH and HbA1c ($r = 0.38$, $p < 0.01$), suggesting that worsening thyroid function is associated with poorer glycemic control. TSH also positively correlated with LDL ($r = 0.39$) and total cholesterol ($r = 0.42$), indicating a link between thyroid suppression and dyslipidemia.

Conversely, FT_4 levels were negatively correlated with FBG ($r = -0.31$, $p = 0.02$), suggesting that higher levels of free thyroxine may improve glucose utilization or insulin sensitivity. These correlations reinforce the hypothesis that thyroid hormones significantly influence both glycemic and lipid regulation.

Table 5. Correlation coefficients between thyroid and metabolic parameters

Correlation Pair	r-value	p-value
TSH vs. HbA1c	0.38	<0.01
TSH vs. LDL	0.39	<0.01
TSH vs. Total Cholesterol	0.42	<0.01
FT_4 vs. Fasting Blood Glucose	-0.31	0.02

Table 6 outlines the results of multivariate regression analysis identifying independent predictors of elevated HbA1c levels. After adjusting for confounding factors such as age, sex, and BMI, TSH remained a significant predictor of glycemic dysregulation ($\beta = 0.26$, $p = 0.01$), further supporting its central role in metabolic homeostasis. BMI ($\beta = 0.34$, $p < 0.01$) and systolic blood pressure ($\beta = 0.28$, $p = 0.02$) were also strong predictors, indicating that adiposity and cardiovascular status contribute to impaired glucose control. Although FT_4 showed a negative association with HbA1c, the relationship was not statistically significant ($p = 0.08$). These findings highlight the multifactorial nature of glycemic regulation and the importance of integrating thyroid assessment in metabolic evaluations.

Table 6. Multivariate regression analysis for predictors of elevated HbA1c

Variable	β Coefficient	p-value
TSH	0.26	0.01
BMI	0.34	<0.01
Systolic Blood Pressure	0.28	0.02
Age	0.12	0.07
FT_4	-0.15	0.08

4. Discussion

The findings reveal that thyroid abnormalities—particularly hypothyroidism—are closely linked with adverse glycemic and lipid profiles, supporting the interdependent relationship between thyroid hormones and metabolic homeostasis. The results demonstrated significantly elevated fasting blood glucose, postprandial glucose, and HbA1c levels among hypothyroid participants compared to euthyroid individuals. Specifically, the hypothyroid group had an average HbA1c of 7.4%, while euthyroid participants had a mean of 6.5% ($p < 0.01$). These findings align with growing evidence that hypothyroidism contributes to insulin resistance and impaired glucose disposal, likely through reduced GLUT4 activity and altered hepatic glucose production[17]. Furthermore, hyperthyroid patients showed moderately elevated fasting glucose levels despite lower HbA1c values, suggesting enhanced hepatic gluconeogenesis coupled with shortened red cell lifespan, which may artifactually lower HbA1c[18].

This hormonal influence on glucose regulation is further supported by correlation analysis in our study, where TSH showed a moderate positive correlation with HbA1c ($r = 0.38$), indicating that worsening thyroid function may be associated with deteriorating glycemic control. Conversely, FT_4 showed a negative correlation with FBG ($r = -0.31$), consistent with studies demonstrating that thyroxine enhances insulin sensitivity and

peripheral glucose uptake[19]. Similar trends have been observed in populations with both overt and subclinical thyroid disorders, suggesting that even subtle thyroid dysfunction may unmask or exacerbate underlying glucose intolerance[20,21].

The present study also found a significant association between thyroid function and lipid profiles. Hypothyroid individuals had markedly elevated total cholesterol and LDL levels ($p < 0.05$), along with reduced HDL cholesterol, underscoring the atherogenic risk posed by thyroid insufficiency. These effects are likely mediated by decreased LDL receptor activity and impaired hepatic clearance of lipoproteins in hypothyroidism[22]. Hyperthyroid patients also exhibited elevated triglycerides, likely due to enhanced lipolysis and hepatic VLDL production[23].

Correlational analysis revealed strong positive relationships between TSH and both LDL ($r = 0.39$) and total cholesterol ($r = 0.42$). These findings are in line with prior reports that subclinical hypothyroidism is independently associated with dyslipidemia, even in the absence of overt symptoms. Such lipid abnormalities contribute to heightened cardiovascular risk in patients with untreated or inadequately managed thyroid disease, especially when compounded by hyperglycemia[24].

Multivariate regression identified TSH as an independent predictor of elevated HbA1c, even after adjusting for confounding factors such as age, sex, and BMI. In addition, BMI and systolic blood pressure emerged as significant contributors to glycemic dysregulation[25]. These findings reinforce the notion that metabolic control is governed by an interplay of hormonal, anthropometric, and cardiovascular factors[26]. Notably, FT_4 showed a negative association with HbA1c in the model, although it did not reach statistical significance—possibly due to sample size limitations or compensatory mechanisms in thyroid homeostasis[27,28].

The observed interplay between thyroid and metabolic parameters in our cohort mirrors findings from other cross-sectional and epidemiological studies, where elevated TSH levels were consistently linked to increased insulin resistance, hyperglycemia, and adverse lipid profiles[29]. Several reports from endocrine-focused populations have confirmed that even subclinical hypothyroidism is associated with elevated HbA1c and fasting glucose, supporting the need for thyroid screening in diabetic and prediabetic patients. Moreover, longitudinal studies have suggested that restoring euthyroid status through levothyroxine therapy may improve insulin sensitivity and lipid levels, further validating the clinical relevance of our findings[30-32].

Interestingly, the metabolic disturbances in hyperthyroid states have shown mixed results across studies—some demonstrating worsened glucose tolerance, while others observe mild improvements in insulin sensitivity due to increased basal metabolic rate. Our study contributes to this dialogue by showing elevated FBG in hyperthyroidism despite relatively preserved HbA1c, highlighting the importance of nuanced interpretation of glycemic markers in such conditions[33].

Given the high burden of both thyroid and metabolic disorders in South Asia, especially among urban populations, our findings underscore the importance of integrated endocrine and metabolic screening. Patients with abnormal thyroid profiles—particularly hypothyroid individuals—should be closely monitored for diabetes and dyslipidemia. Conversely, patients with poorly controlled diabetes may benefit from thyroid function testing to detect subclinical dysfunctions that may be impeding metabolic control[34,35].

Moreover, the strong predictive value of BMI and systolic blood pressure for glycemic abnormalities supports lifestyle-based interventions aimed at weight reduction and blood pressure control as adjuncts to endocrine management. These strategies are particularly vital in resource-constrained settings where healthcare access may be limited.

5. Conclusion

In summary, this study highlights a significant relationship between thyroid dysfunction—particularly hypothyroidism—and metabolic derangements including hyperglycemia and dyslipidemia. TSH emerged as a key hormonal predictor of elevated HbA1c, reinforcing the metabolic role of thyroid hormones. These findings support routine thyroid screening in patients with diabetes or metabolic syndrome and advocate for a multidisciplinary approach to managing endocrine and metabolic disorders in clinical practice.

6. Limitations of the study

While this study provides valuable insights into the relationship between thyroid dysfunction and metabolic dysregulation, several limitations should be acknowledged:

1. Cross-Sectional Design: As a cross-sectional study, the findings demonstrate associations but cannot establish causality. It is unclear whether thyroid dysfunction directly contributes to metabolic abnormalities or vice versa.
2. Single-Center Study: The study was conducted at a single diabetic care center in Dhaka, which may limit the generalizability of the results to other populations or healthcare settings, especially rural or non-diabetic centers.
3. Convenience Sampling: Participants were selected using a non-randomized convenience sampling method, which may introduce selection bias and affect the representativeness of the sample.
4. Lack of Insulin Resistance Assessment: Important indicators such as fasting insulin levels and HOMA-IR (Homeostatic Model Assessment of Insulin Resistance) were not included, limiting the ability to assess insulin sensitivity and resistance directly.
5. Uncontrolled Confounders: Although adjustments were made for age, sex, BMI, and blood pressure, other potential confounders—such as dietary habits, physical activity levels, smoking status, and socioeconomic factors—were not controlled for.
6. Subclinical Thyroid Dysfunction Grouping: Both subclinical and overt thyroid dysfunction cases were combined within the hypothyroid and hyperthyroid categories, which may obscure differences in metabolic impact between subclinical and overt forms.
7. Absence of Longitudinal Follow-Up: The study lacked follow-up data to assess the effect of thyroid treatment or metabolic intervention over time, which could provide stronger evidence of causality and progression.
8. Sample Size for Subgroups: The relatively small number of participants in the hyperthyroid group (n = 16) may limit statistical power and the ability to detect more subtle differences in this subgroup.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Author contributions

All authors contributed equally to the conception, design, data collection, analysis, and writing of this manuscript. All authors have read and approved the final version.

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