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# Comparative Evaluation of Thyroid Function Profile Among Patients with Type 2 Diabetes Mellitus, Hypothyroidism, and Their Coexistence: A Hospital-Based Cross-Sectional Comparative Study

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## ABSTRACT

### Background

Type 2 Diabetes Mellitus (T2DM) and hypothyroidism are among the most prevalent endocrine disorders worldwide and frequently coexist because of their shared metabolic and hormonal interactions. Thyroid hormones play a pivotal role in regulating carbohydrate metabolism, insulin secretion, and peripheral insulin sensitivity. Thyroid dysfunction in patients with T2DM may adversely affect glycemic control and increase the risk of cardiovascular and metabolic complications. Early identification of thyroid abnormalities may therefore contribute to improved clinical management and better long-term outcomes.

### Aim

To evaluate the thyroid function profile among patients with Type 2 Diabetes Mellitus and compare thyroid hormone levels across healthy controls, patients with hypothyroidism, and patients with coexisting Type 2 Diabetes Mellitus and hypothyroidism.

### Materials and Methods

This hospital-based cross-sectional comparative study was conducted in the Department of Biochemistry in collaboration with the Department of General Medicine at a tertiary care teaching

hospital. A total of **200 male participants** aged **20–65 years** were enrolled and equally distributed into four groups: healthy controls (n=50), patients with Type 2 Diabetes Mellitus (n=50), patients with hypothyroidism (n=50), and patients with both Type 2 Diabetes Mellitus and hypothyroidism (n=50). Fasting Blood Sugar (FBS), Post-Prandial Blood Sugar (PPBS), Glycated Hemoglobin (HbA1c), Thyroid-Stimulating Hormone (TSH), Free Triiodothyronine (FT3), and Free Thyroxine (FT4) were estimated using standardized laboratory methods. Statistical analysis was performed using one-way Analysis of Variance (ANOVA) followed by Tukey's post hoc test. A p-value of <0.05 was considered statistically significant.

**Results**

The mean age of the participants was comparable among the study groups (**p=0.7021**). Patients with T2DM and those with coexisting T2DM and hypothyroidism demonstrated significantly higher FBS, PPBS, and HbA1c levels than healthy controls and patients with hypothyroidism alone (**p<0.0001**). Serum TSH concentrations were significantly elevated in patients with hypothyroidism (**27.00 ± 13.81 µIU/mL**) and in those with coexisting T2DM and hypothyroidism (**35.27 ± 18.55 µIU/mL**) compared with controls (**3.37 ± 1.40 µIU/mL**) (**p<0.0001**). Conversely, FT3 and FT4 concentrations were significantly reduced in hypothyroid groups (**p<0.0001**), indicating marked thyroid hormone abnormalities, particularly in patients with coexisting endocrine disorders.

**Conclusion**

Thyroid dysfunction, particularly hypothyroidism, is strongly associated with Type 2 Diabetes Mellitus and is accompanied by significant alterations in glycemic and thyroid function parameters. Patients with coexisting diabetes and hypothyroidism exhibit greater metabolic derangements than those with either disorder alone. Routine assessment of thyroid function in patients with T2DM may facilitate early diagnosis, optimize metabolic control, and potentially reduce the risk of long-term cardiovascular complications.

**Keywords**

Type 2 Diabetes Mellitus; Hypothyroidism; Thyroid Function Tests; Thyroid-Stimulating Hormone; Hemoglobin A1c; Insulin Resistance.

## INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction. It accounts for nearly 90–95% of all cases of diabetes mellitus and represents one of the fastest-growing global health challenges. According to the International Diabetes Federation (IDF), the prevalence of diabetes continues to rise worldwide, with India bearing a substantial proportion of the global disease burden. Chronic hyperglycemia predisposes affected individuals to both microvascular complications, including retinopathy, nephropathy, and neuropathy, and macrovascular complications such as coronary artery disease, cerebrovascular disease, and peripheral arterial disease, contributing significantly to morbidity, mortality, and healthcare expenditure.[14],[15]

Thyroid disorders are the second most common endocrine disorders after diabetes mellitus and frequently coexist with T2DM. The physiological relationship between thyroid hormones and glucose metabolism is complex and bidirectional. Thyroid hormones regulate carbohydrate, lipid, and protein metabolism by influencing hepatic glucose production, intestinal glucose absorption, pancreatic β-cell function, insulin secretion, and peripheral glucose utilization. Consequently, alterations in thyroid hormone concentrations can disrupt metabolic homeostasis, impair insulin sensitivity, and adversely affect glycemic control.[1,2,8]

Hypothyroidism, particularly subclinical hypothyroidism, is the most commonly reported thyroid abnormality among patients with T2DM. Reduced thyroid hormone activity decreases basal metabolic rate, impairs insulin-mediated glucose uptake, and promotes insulin resistance, thereby contributing to poor glycemic control. In addition, hypothyroidism is associated with dyslipidemia, endothelial dysfunction, oxidative stress, chronic low-grade inflammation, and accelerated atherosclerosis, all of which increase the risk of cardiovascular disease. Conversely, hyperthyroidism enhances hepatic glucose production and insulin degradation, leading to worsening hyperglycemia and increased insulin requirements.[1,8,10]

Several epidemiological studies have reported a higher prevalence of thyroid dysfunction among patients with T2DM than in the general population, with estimates ranging from 10% to 30%, depending on the study population and diagnostic criteria.[3,4,24,29] Because thyroid dysfunction often remains clinically silent during its early stages, it may go undiagnosed unless actively investigated. Undetected thyroid abnormalities may further impair metabolic control and contribute to the progression of both microvascular and macrovascular diabetic complications.[20,21]

The coexistence of diabetes mellitus and thyroid dysfunction has important clinical implications. Both disorders independently contribute to insulin resistance, abnormalities in lipid metabolism, endothelial dysfunction, and chronic inflammation. Their coexistence may therefore exert additive or synergistic effects, increasing the risk of cardiovascular disease and other diabetes-related complications. Previous studies have demonstrated that patients with T2DM and hypothyroidism have a higher prevalence of dyslipidemia, diabetic nephropathy, retinopathy, and cardiovascular morbidity than patients with diabetes alone.[31–38] These observations emphasize the importance of evaluating thyroid function as part of the comprehensive assessment of patients with T2DM.

Despite the growing body of evidence supporting the association between thyroid dysfunction and T2DM, routine thyroid function assessment is not consistently performed in clinical practice, particularly in resource-limited settings. Furthermore, data from the Indian population remain limited, and regional variations in the prevalence and pattern of thyroid dysfunction warrant further investigation. Identifying thyroid abnormalities at an early stage may facilitate timely intervention, improve glycemic control, optimize metabolic outcomes, and potentially reduce the burden of long-term diabetic complications.

**Therefore, the present study was undertaken to evaluate the thyroid function profile among patients with Type 2 Diabetes Mellitus and to compare thyroid hormone levels across healthy controls, patients with hypothyroidism, and patients with coexisting Type 2 Diabetes Mellitus and hypothyroidism. The study also aimed to examine the relationship between thyroid dysfunction and glycemic status in order to highlight the clinical importance of routine thyroid function assessment in patients with T2DM.**

## MATERIALS AND METHODS

## Study Design and Setting

This hospital-based cross-sectional comparative study was conducted in the Department of Biochemistry in collaboration with the Department of General Medicine at Jaipur National University Institute for Medical Sciences and Research Centre (JNUIMSRC), Jaipur, Rajasthan, India. The study was carried out over a period of one year after obtaining approval from the Institutional Ethics Committee.

## Ethical Approval

The study protocol was approved by the Institutional Ethics Committee of Jaipur National University Institute for Medical Sciences and Research Centre, Jaipur (Approval No. **JNUIMSRC/IEC/2023/91**, dated **31 July 2023**). All procedures were performed in accordance with the ethical principles of the Declaration of Helsinki (2013 revision). Written informed consent was obtained from all participants before enrolment.

## Study Population

A total of **200 male participants** aged **20–65 years** were recruited using a convenient sampling technique and equally allocated into four study groups (n=50 each):

- **Group I (Control):** Apparently healthy, age-matched individuals without diabetes mellitus or thyroid dysfunction.
- **Group II (Type 2 Diabetes Mellitus):** Patients with previously diagnosed Type 2 Diabetes Mellitus.
- **Group III (Hypothyroidism):** Patients with clinically and biochemically confirmed hypothyroidism.
- **Group IV (Type 2 Diabetes Mellitus with Hypothyroidism):** Patients diagnosed with both Type 2 Diabetes Mellitus and hypothyroidism.

The diagnosis of Type 2 Diabetes Mellitus was established according to the **American Diabetes Association (ADA) diagnostic criteria**, while hypothyroidism was diagnosed based on clinical findings together with biochemical evidence of elevated serum thyroid-stimulating hormone (TSH) and reduced free thyroxine (FT4), or documented medical records.

## Inclusion Criteria

Participants fulfilling the following criteria were included in the study:

- Male participants aged between **20 and 65 years**.
- Patients with a confirmed diagnosis of Type 2 Diabetes Mellitus.
- Patients with a confirmed diagnosis of hypothyroidism.
- Patients with coexisting Type 2 Diabetes Mellitus and hypothyroidism.
- Apparently healthy volunteers without known endocrine disorders for the control group.
- Individuals willing to participate and who provided written informed consent.

## Exclusion Criteria

Participants meeting any of the following criteria were excluded:

- Type 1 Diabetes Mellitus.
- Hyperthyroidism or other thyroid disorders apart from hypothyroidism.
- Chronic liver disease, chronic kidney disease, malignancy, autoimmune disorders, or other major systemic illnesses.
- Acute infections or inflammatory disorders.
- Pregnancy or lactation.
- Patients receiving medications known to interfere with thyroid function tests, including corticosteroids, amiodarone, lithium, or antithyroid drugs.
- Individuals unwilling to participate in the study.

**Sample Collection**

After an overnight fast of **8–12 hours**, approximately **5 mL of venous blood** was collected from the antecubital vein under strict aseptic precautions. Blood samples for plasma glucose estimation were collected in fluoride-oxalate tubes, while EDTA-anticoagulated blood was used for HbA1c estimation. Serum was separated by centrifugation and used for thyroid hormone analysis.

**Biochemical Analysis**

The following biochemical parameters were estimated in all participants:

- Fasting Blood Sugar (FBS)
- Post-Prandial Blood Sugar (PPBS)
- Glycated Hemoglobin (HbA1c)
- Thyroid-Stimulating Hormone (TSH)
- Free Triiodothyronine (FT3)
- Free Thyroxine (FT4)

Fasting and post-prandial plasma glucose levels were estimated using the **glucose oxidase-peroxidase (GOD-POD) enzymatic method**. Glycated hemoglobin (HbA1c) was measured using a standardized method employed in the institutional laboratory. Serum TSH, FT3, and FT4 concentrations were estimated using a **fully automated chemiluminescent immunoassay (CLIA)** system following the manufacturer's instructions. Internal quality control and external quality assurance procedures were routinely followed to ensure the reliability and accuracy of laboratory results.

**Statistical Analysis**

Data were entered into Microsoft Excel and analyzed using **IBM Statistical Package for the Social Sciences (SPSS) Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA)**. Continuous variables were expressed as **mean ± standard deviation (Mean ± SD)**. Comparisons among the four study groups were performed using **one-way Analysis of Variance (ANOVA)**, followed by **Tukey's post hoc multiple comparison test** to identify intergroup differences. A **p-value <0.05** was considered statistically significant.

**RESULTS**

A total of **200 male participants** were included in the study and equally distributed into four groups: healthy controls, patients with Type 2 Diabetes Mellitus (T2DM), patients with hypothyroidism, and patients with coexisting T2DM and hypothyroidism (n=50 each).

**Baseline Characteristics**

The mean age of the study participants ranged from **41.7 ± 8.8** to **44.0 ± 10.3 years** across the four study groups. There was **no statistically significant difference** in age distribution among the groups (One-way ANOVA, **p=0.7021**), indicating that the participants were comparable with respect to age (**Table 1; Figure 1**).

**Glycemic Parameters**

Comparison of glycemic parameters demonstrated significant differences among the four study groups (**Table 2**).

The mean fasting blood sugar (FBS) was significantly higher in patients with **T2DM (161.1 ± 73.87 mg/dL)** and **T2DM with hypothyroidism (135.9 ± 16.45 mg/dL)** compared with healthy controls (**84.07 ± 12.82 mg/dL**) and patients with hypothyroidism alone (**89.27 ± 19.29 mg/dL**) (**p<0.0001**).

Similarly, post-prandial blood sugar (PPBS) levels were markedly elevated in diabetic participants, with mean values of **346.9 ± 80.10 mg/dL** in the T2DM group and **329.9 ± 68.16 mg/dL** in the T2DM with hypothyroidism group. In contrast, healthy controls and hypothyroid participants exhibited comparatively lower PPBS values (**123.5 ± 16.89 mg/dL** and **129.6 ± 21.16 mg/dL**, respectively). The observed differences were highly statistically significant (**p<0.0001**).

Assessment of long-term glycemic control showed a similar trend. The mean HbA1c was **7.53 ± 1.91%** among patients with T2DM and **6.92 ± 1.08%** in those with coexisting T2DM and hypothyroidism, compared with **5.16 ± 0.80%** in healthy controls and **5.58 ± 0.97%** in patients with hypothyroidism (**p<0.0001**).

Overall, patients with diabetes exhibited significantly poorer glycemic control than non-diabetic participants, while the coexistence of hypothyroidism appeared to further aggravate metabolic

**Thyroid Function Profile**

Significant differences in thyroid function parameters were observed among the study groups (**Table 3**).

Serum thyroid-stimulating hormone (TSH) concentrations progressively increased from healthy controls (**3.37 ± 1.40 µIU/mL**) to patients with T2DM (**5.43 ± 2.25 µIU/mL**), hypothyroidism (**27.00 ± 13.81 µIU/mL**), and coexisting T2DM with hypothyroidism (**35.27 ± 18.55 µIU/mL**). The highest TSH levels were observed in patients with both endocrine disorders (**p<0.0001**).

Conversely, serum FT3 concentrations were significantly lower among hypothyroid participants. The mean FT3 concentration was **2.95 ± 0.54 pg/mL** in healthy controls and **2.85 ± 0.97 pg/mL** in diabetic participants, whereas patients with hypothyroidism and those with coexisting T2DM and hypothyroidism demonstrated significantly lower concentrations (**1.82 ± 0.44 pg/mL** and **2.24 ± 0.76 pg/mL**, respectively) (**p<0.0001**).

Similarly, serum FT4 concentrations were significantly reduced in patients with hypothyroidism. The lowest FT4 values were recorded in the hypothyroidism (**0.80 ± 0.49 ng/dL**) and T2DM with hypothyroidism (**0.87 ± 0.55 ng/dL**) groups compared with healthy controls (**1.55 ± 0.35 ng/dL**) and diabetic patients (**1.23 ± 0.54 ng/dL**) (**p<0.0001**).

Collectively, these findings demonstrate that thyroid dysfunction was characterized by significantly elevated TSH levels accompanied by reduced FT3 and FT4 concentrations, with the most pronounced hormonal alterations observed in patients with coexisting Type 2 Diabetes Mellitus and hypothyroidism (**Table 3; Figure 3A–C**).

## DISCUSSION

The present study provides further evidence of the close association between Type 2 Diabetes Mellitus (T2DM) and thyroid dysfunction, particularly hypothyroidism. Patients with coexisting T2DM and hypothyroidism demonstrated more pronounced abnormalities in both glycemic indices and thyroid hormone profile than those with either disorder alone. These findings emphasize the intricate interplay between thyroid hormones and glucose metabolism and suggest that thyroid dysfunction may adversely influence metabolic control in individuals with diabetes.

The absence of a significant difference in age distribution among the four study groups indicates that the observed biochemical variations were unlikely to be influenced by age. This enhances the internal validity of the study and permits more reliable comparison of thyroid function and glycemic parameters across the study groups. Comparable demographic characteristics have also been reported in previous studies evaluating thyroid dysfunction among patients with diabetes.[3,5,22]

A key observation in the present study was the significantly higher FBS, PPBS, and HbA1c levels among patients with T2DM, particularly those with concomitant hypothyroidism. Although patients with isolated hypothyroidism maintained relatively normal glucose concentrations, the coexistence of diabetes and thyroid dysfunction was associated with poorer glycemic control. This finding supports the concept that thyroid hormone deficiency can aggravate disturbances in glucose metabolism and make diabetes more difficult to control. Similar observations have been reported by **Biondi et al.** and **Duntas et al.**, who demonstrated that thyroid hormones play an essential role in maintaining glucose homeostasis by regulating insulin secretion, hepatic glucose production, and peripheral insulin sensitivity.[1,2]

The underlying biological mechanisms are well recognized. Reduced thyroid hormone activity decreases glucose uptake by skeletal muscle and adipose tissue, impairs insulin-mediated glucose utilization, and alters hepatic glucose metabolism, thereby promoting insulin resistance. In addition, hypothyroidism is associated with reduced expression of glucose transporter proteins, diminished energy expenditure, and altered lipid metabolism, all of which contribute to metabolic deterioration. These mechanisms are consistent with the observations of **Maratou et al.** and **Dimitriadis et al.**, who demonstrated impaired insulin sensitivity in patients with hypothyroidism.[16,17] The higher HbA1c levels observed in patients with coexisting diabetes and hypothyroidism in the present study are therefore biologically plausible.

Another important finding was the marked elevation in serum TSH levels accompanied by significant reductions in FT3 and FT4 concentrations among hypothyroid patients, particularly those with coexisting T2DM. These hormonal changes reflect the expected biochemical pattern of hypothyroidism but also suggest that diabetes may amplify endocrine dysfunction. Similar findings have been reported by **Palma et al.**, who observed a greater prevalence of thyroid abnormalities among diabetic patients than in the general population.[3] Likewise, **Udiong et al.** reported altered thyroid hormone profiles in patients with T2DM and emphasized the importance of routine thyroid function testing in this population.[5]

The coexistence of diabetes mellitus and hypothyroidism represents a metabolically unfavorable state in which each disorder appears to intensify the effects of the other. While hypothyroidism contributes to reduced metabolic activity, dyslipidemia, endothelial dysfunction, oxidative stress, and chronic inflammation, diabetes further worsens insulin resistance and vascular injury through persistent hyperglycemia. The combined effect of these abnormalities may explain the more severe biochemical disturbances observed in the present study. This interpretation is supported by the systematic review conducted by **Han et al.**, which identified subclinical hypothyroidism as a frequent metabolic comorbidity in patients with T2DM.[4] **Chaker et al.** also emphasized that even mild alterations in thyroid hormone status can significantly influence carbohydrate and lipid metabolism.[8]

The cardiovascular implications of these findings deserve particular attention. Both diabetes mellitus and hypothyroidism are independently associated with dyslipidemia, endothelial dysfunction, arterial stiffness, and accelerated atherosclerosis. Consequently, patients with both disorders are likely to have an even greater cardiovascular risk. **Jabbar et al.** demonstrated that thyroid hormones play an essential role in maintaining cardiovascular function and that thyroid dysfunction is associated with an increased risk of coronary artery disease, heart failure, arrhythmias, and cardiovascular mortality.[31] Similarly, **Rodondi et al.** reported that subclinical hypothyroidism independently increases the risk of coronary heart disease and cardiovascular death, particularly in individuals with persistently elevated TSH concentrations.[33] These observations highlight the potential value of thyroid function assessment as part of cardiovascular risk evaluation in patients with T2DM.

Emerging evidence also suggests that thyroid dysfunction may contribute to the progression of diabetic microvascular complications. **Chen et al.** demonstrated an association between subclinical hypothyroidism and diabetic nephropathy, while **Kim et al.** reported a higher prevalence of severe diabetic retinopathy among patients with hypothyroidism.[20,21] Although the present study did not evaluate diabetic complications directly, the observed thyroid hormone abnormalities reinforce the need for comprehensive endocrine assessment in patients with diabetes.

The present findings are also consistent with reports from India. **Gutch et al.**, **Singh et al.**, and **Pasupathi et al.** similarly documented a higher frequency of thyroid dysfunction among patients with T2DM and emphasized the importance of routine thyroid function screening for improving metabolic control and facilitating early intervention.[22,23,27] The consistency of these findings across different populations strengthens the evidence supporting routine thyroid evaluation in patients with diabetes.

One of the strengths of this study is the inclusion of four well-defined comparison groups, which allowed simultaneous evaluation of glycemic and thyroid function parameters under different clinical conditions. The use of standardized laboratory methods and uniform statistical analysis further enhances the reliability of the findings. Nevertheless, certain limitations should be considered. The study was conducted at a single tertiary care center with a relatively modest sample size and included only male participants, which may limit the generalizability of the findings. In addition, the cross-sectional design does not permit causal inference, and parameters such as thyroid autoantibodies, lipid profile, inflammatory biomarkers, and long-term clinical outcomes were not evaluated. Future multicenter prospective studies are therefore needed to clarify the causal relationship between thyroid dysfunction and diabetes and to determine whether early treatment of thyroid abnormalities improves long-term metabolic and cardiovascular outcomes.

Overall, the findings of the present study reinforce the close association between thyroid dysfunction and Type 2 Diabetes Mellitus. The greater hormonal abnormalities and poorer glycemic control

observed in patients with coexisting diabetes and hypothyroidism highlight the importance of integrating thyroid function assessment into routine diabetes care. Early identification and appropriate management of thyroid dysfunction may improve metabolic control, reduce cardiovascular risk, and ultimately contribute to better long-term clinical outcomes.

## CONCLUSION

The present study demonstrated a significant association between Type 2 Diabetes Mellitus (T2DM) and thyroid dysfunction, particularly hypothyroidism. Patients with coexisting T2DM and hypothyroidism exhibited significantly elevated serum TSH levels, reduced FT3 and FT4 concentrations, and poorer glycemic control, as reflected by higher fasting blood glucose, post-prandial blood glucose, and HbA1c levels, compared with healthy controls and patients with either disorder alone.

These findings highlight the close interplay between thyroid hormones and glucose metabolism and suggest that thyroid dysfunction may contribute to worsening metabolic status in patients with T2DM. Since thyroid abnormalities are often asymptomatic during the early stages, routine assessment of thyroid function, particularly serum TSH, FT3, and FT4, may facilitate early diagnosis and timely therapeutic intervention. Such an approach has the potential to improve glycemic control, optimize metabolic outcomes, and reduce the risk of long-term cardiovascular complications.

Further multicenter prospective studies involving larger and more diverse populations are warranted to clarify the causal relationship between thyroid dysfunction and T2DM and to evaluate the long-term impact of thyroid abnormalities on diabetic complications.

## CLINICAL SIGNIFICANCE

The findings of the present study emphasize the clinical importance of routine thyroid function assessment in patients with Type 2 Diabetes Mellitus, particularly those with poor glycemic control or clinical features suggestive of thyroid dysfunction. Early identification and appropriate management of thyroid abnormalities may improve metabolic control, facilitate individualized treatment strategies, and potentially reduce the risk of diabetes-related cardiovascular and microvascular complications.

## LIMITATIONS

The present study has certain limitations. First, it was conducted at a single tertiary care center, which may limit the generalizability of the findings. Second, the cross-sectional study design precludes establishing a causal relationship between thyroid dysfunction and Type 2 Diabetes Mellitus. Third, only male participants were included, thereby limiting extrapolation of the findings to the general population. In addition, the relatively modest sample size and the absence of lipid profile, thyroid autoantibody, inflammatory biomarker, and long-term follow-up data restricted a more comprehensive evaluation of the metabolic and cardiovascular implications of thyroid dysfunction in diabetic patients.

## CONFLICT OF INTEREST

The authors declare that there are **no conflicts of interest** related to this study.

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## AUTHOR CONTRIBUTIONS

**Kishor Kumar:** Conceptualization, data collection, laboratory investigations, formal analysis, statistical analysis, manuscript drafting, and literature review.

**Rubal Singh:** Data acquisition, data curation, literature review, interpretation of results, critical revision of the manuscript for important intellectual content, and final approval of the manuscript.

M. **Itagappa:** Study design, supervision, interpretation of data, critical review of the manuscript, and overall academic guidance.

**Mandayal Jamatia:** Conceptualization, methodology, supervision, validation of results, manuscript editing, critical revision, and final approval of the manuscript.

**All authors** contributed substantially to the study, reviewed and approved the final version of the manuscript, and agree to be accountable for all aspects of the work.

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**Table 1. Baseline Characteristics of the Study Population According to the Study Groups**

| **Study Group** | **Mean Age (Years)** |
|---|---|
| Control | 41.7 ± 8.8 |
| Type 2 Diabetes Mellitus | 42.3 ± 10.1 |
| Hypothyroidism | 44.0 ± 10.3 |
| Type 2 Diabetes Mellitus with Hypothyroidism | 42.4 ± 10.8 |

**Table 1** presents the baseline age distribution of the study participants. The mean age was comparable among the four study groups, and no statistically significant difference was observed (One-way ANOVA, **p = 0.7021**), indicating that the study groups were well matched with respect to age.

**Table 2. Comparison of Glycemic Parameters Among the Study Groups**

| Parameter | Control | Type 2 Diabetes Mellitus | Hypothyroidism | Type 2 Diabetes Mellitus with Hypothyroidism | p-value |
|---|---|---|---|---|---|
| FBS (mg/dL) | 84.07 ± 12.82 | 161.1 ± 73.87 | 89.27 ± 19.29 | 135.9 ± 16.45 | <0.0001 |
| PPBS (mg/dL) | 123.5 ± 16.89 | 346.9 ± 80.10 | 129.6 ± 21.16 | 329.9 ± 68.16 | <0.0001 |
| HbA1c (%) | 5.16 ± 0.80 | 7.53 ± 1.91 | 5.58 ± 0.97 | 6.92 ± 1.08 | <0.0001 |

**Table 2** summarizes the comparison of glycemic parameters among the study groups. Patients with Type 2 Diabetes Mellitus, with or without hypothyroidism, exhibited significantly higher fasting blood sugar (FBS), post-prandial blood sugar (PPBS), and glycated hemoglobin (HbA1c) levels than healthy controls and patients with hypothyroidism alone. The differences among the groups were highly statistically significant (**$p < 0.0001$**).

**Table 3. Comparison of Thyroid Function Parameters Among the Study Groups**

| Parameter | Control | Type 2 Diabetes Mellitus | Hypothyroidism | Type 2 Diabetes Mellitus with Hypothyroidism | p-value |
|---|---|---|---|---|---|
| TSH (μIU/mL) | 3.37 ± 1.40 | 5.43 ± 2.25 | 27.00 ± 13.81 | 35.27 ± 18.55 | <0.0001 |
| FT3 (pg/mL) | 2.95 ± 0.54 | 2.85 ± 0.97 | 1.82 ± 0.44 | 2.24 ± 0.76 | <0.0001 |
| FT4 (ng/dL) | 1.55 ± 0.35 | 1.23 ± 0.54 | 0.80 ± 0.49 | 0.87 ± 0.55 | <0.0001 |

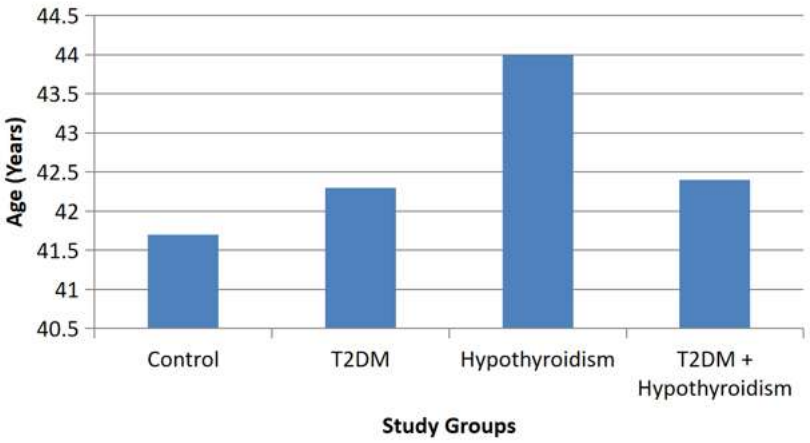
**Table 3** shows the comparison of thyroid function parameters among the study groups. Serum thyroid-stimulating hormone (TSH) levels were significantly elevated, whereas free triiodothyronine (FT3) and free thyroxine (FT4) concentrations were significantly reduced in patients with hypothyroidism and those with coexisting Type 2 Diabetes Mellitus and hypothyroidism. The most

pronounced thyroid hormone abnormalities were observed in the combined disease group, and all differences were statistically significant (**$p < 0.0001$**).

**Values are expressed as Mean ± Standard Deviation (SD). Statistical analysis was performed using one-way Analysis of Variance (ANOVA), followed by Tukey's post hoc test. A p-value <0.05 was considered statistically significant.**

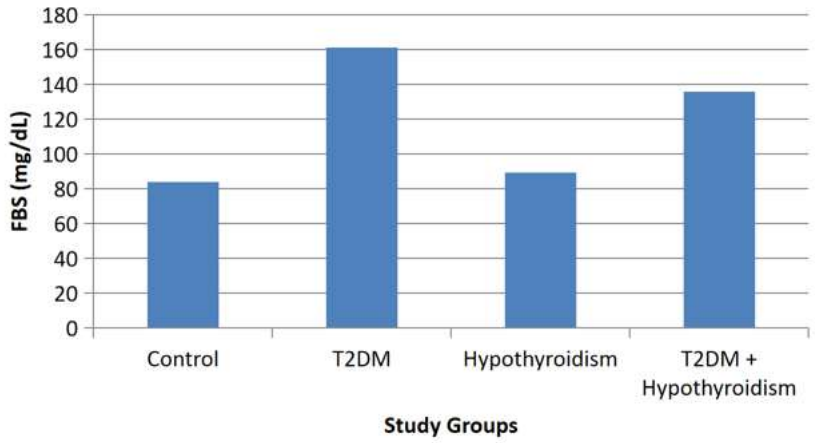
**Abbreviations:** FBS, Fasting Blood Sugar; PPBS, Post-Prandial Blood Sugar; HbA1c, Glycated Hemoglobin; TSH, Thyroid-Stimulating Hormone; FT3, Free Triiodothyronine; FT4, Free Thyroxine

**Figure 1: Comparison of the Mean Age of Participants Across the Study Groups.**



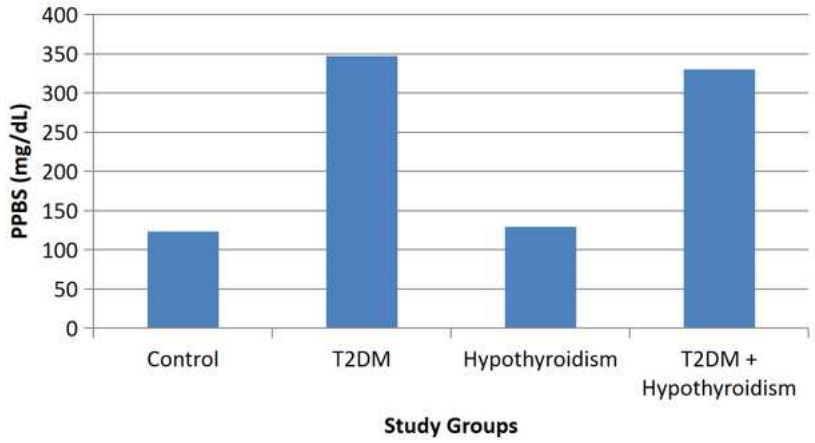
Bar graph illustrating the mean age of participants in the control, Type 2 Diabetes Mellitus (T2DM), hypothyroidism, and T2DM with hypothyroidism groups. No statistically significant difference in age distribution was observed among the study groups ($p$=0.7021).

**Figure 2A: Comparison of Fasting Blood Sugar Levels Among the Study Groups.**



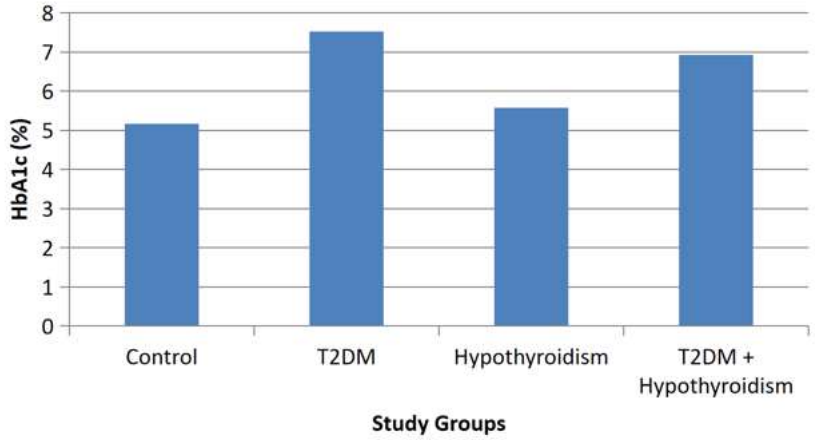

Mean fasting blood sugar (FBS) levels in the four study groups. Patients with T2DM and those with coexisting T2DM and hypothyroidism demonstrated significantly higher FBS levels than controls and hypothyroid subjects ($p$<0.0001).

**Figure 2B: Comparison of Post-Prandial Blood Sugar Levels Among the Study Groups.**



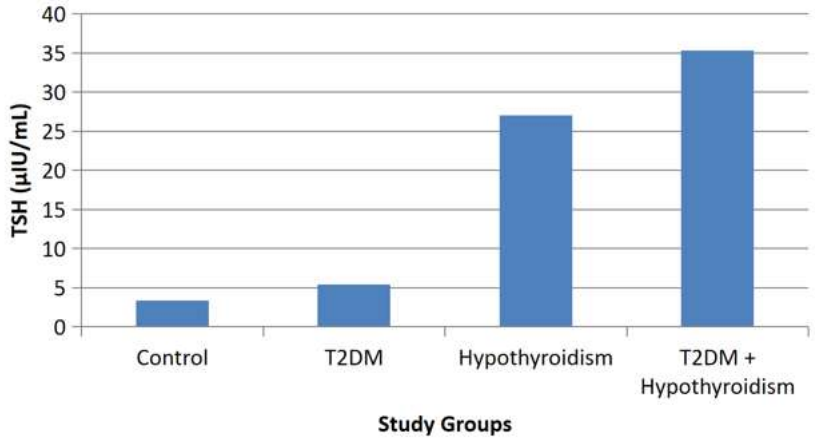

Mean post-prandial blood sugar (PPBS) levels across the four study groups. Significantly elevated PPBS levels were observed in diabetic participants compared with non-diabetic participants ($p$<0.0001).

**Figure 2C: Comparison of Glycated Hemoglobin (HbA1c) Among the Study Groups.**



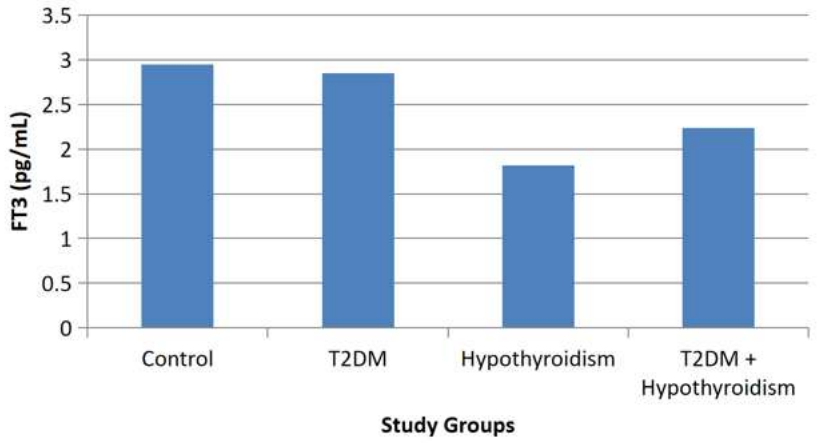

Mean HbA1c levels among the study groups. Patients with T2DM exhibited significantly higher HbA1c values, indicating poorer long-term glycemic control ($p$<0.0001).

**Figure 3A: Comparison of Serum Thyroid-Stimulating Hormone Levels Among the Study Groups.**



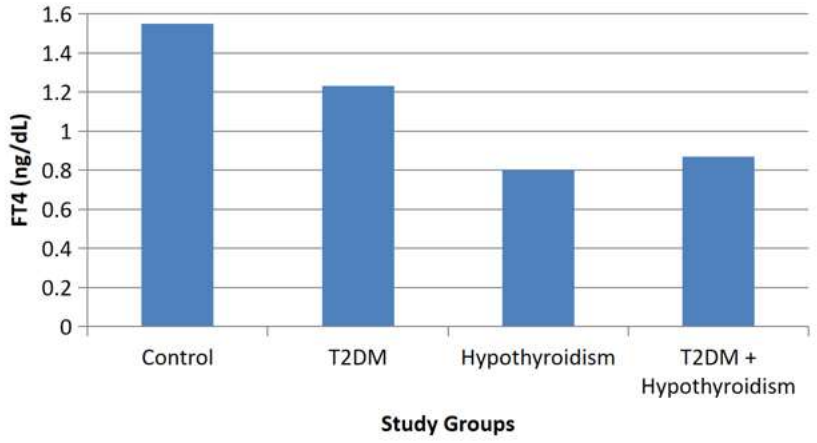
**Legend:** Mean serum TSH concentrations among the study groups. The highest TSH levels were observed in patients with coexisting T2DM and hypothyroidism ($p$<0.0001).

**Figure 3B. Comparison of Serum Free Triiodothyronine Levels Among the Study Groups.**




Mean serum FT3 concentrations among the study groups. FT3 levels were significantly reduced in patients with hypothyroidism, with or without T2DM ($p$<0.0001).

**Figure 3C. Comparison of Serum Free Thyroxine Levels Among the Study Groups.**




Mean serum FT4 concentrations among the study groups. Patients with hypothyroidism demonstrated significantly lower FT4 levels than healthy controls and diabetic patients without hypothyroidism ($p$<0.0001).