STEMedicine

ORIGINAL RESEARCH ARTICLE

Relationship Between Thyroid Dysfunction and Glycaemic Control in Patients with Type 2 Diabetes Mellitus

Dr. Shivaraj Hadimani[1]*, Dr. Pragati Nalkamana[2], Dr. Manish Kumar[3]

[1]Assistant Professor, Department of General Medicine, S.R. Patil Medical College, Badagandi, Bagalkote, Karnataka
[2]House Surgeon, S.S. Institute of Medical Sciences, Davanagere, Karnataka
[3]Assistant Professor, Department of General Medicine, Balvir Singh Tomar Institute of Medical Science Research & Hospital, Jaipur - Delhi Highway, Jaipur, Rajasthan

Corresponding Author: Dr. Shivaraj Hadimani
Email: drshivarajhadimani@gmail.com

Abstract: Background: Thyroid dysfunction commonly coexists with type 2 diabetes mellitus (T2DM), but its relationship with glycaemic control remains uncertain. This study evaluated the prevalence of thyroid dysfunction and its association with glycaemic parameters in patients with T2DM.**Methods:** A total of 350 patients with T2DM were assessed for biochemical thyroid status, HbA1c, fasting glucose, and postprandial glucose. Multivariable regression examined the association between thyroid dysfunction and HbA1c after adjustment for demographic, clinical, and treatment-related factors.**Results:** Thyroid status was classifiable in 349 patients, of whom 89 (25.5%) had thyroid dysfunction, most commonly subclinical hypothyroidism. Mean HbA1c was similar in patients with thyroid dysfunction and euthyroid patients (8.00% vs 7.93%). After adjustment, thyroid dysfunction was associated with an HbA1c difference of 0.03 percentage points (95% CI −0.25 to 0.30). Thyroid dysfunction was also not associated with HbA1c ≥7%. TSH and free T4 were not significantly correlated with HbA1c.**Conclusion:** Thyroid dysfunction was common in T2DM but was not independently associated with poorer glycaemic control.

Keywords: type 2 diabetes mellitus; thyroid dysfunction; HbA1c; subclinical hypothyroidism; glycaemic control

Received:10.04.2026 | Revised:25.04.2026 | Accepted:05.05.2026 | Published:01.06.2026

Citation: Dr. Shivaraj Hadimani *et al* (2026). Relationship Between Thyroid Dysfunction and Glycaemic Control in Patients with Type 2 Diabetes Mellitus. STEMed, 7(1): 52-58.

Introduction

Thyroid disorders are common in patients with type 2 diabetes mellitus (T2DM) and can interact with T2DM via multiple metabolic pathways. Diabetes-related metabolic disturbances can affect thyroid function, and thyroid hormones can affect glucose production in the liver, glucose absorption in the intestine, function of pancreatic β-cells, insulin sensitivity, and glucose utilization in the periphery [1]. This bi-directional association has led to growing interest in thyroid abnormalities as possibly relevant co-morbidities in patients with T2DM.

Thyroid dysfunction seems to be more common in people with T2DM than in the general population. A recent systematic review and meta-analysis estimated the overall prevalence to be around 20%, with subclinical hypothyroidism being the most common abnormality [2]. The prevalence rates in individual clinical studies have been reported to be between about 16% and 27%, and are higher in older adults, women and those with less metabolic control [3,4]. Subclinical hypothyroidism is common and can be clinically undiagnosed due to vague symptoms or being attributed to diabetes and

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

associated comorbidities.

The association between thyroid status and glycaemic control, however, is unclear. In some studies, thyroid dysfunction has been associated with increased HbA1c and fasting glucose levels [3,5] while in others, the relationship was not as strong or consistent after controlling for clinical and metabolic parameters [6]. Thyroid hormones may influence glucose homeostasis via insulin action and energy metabolism, which may explain a link with glycaemic control [1]. Thyroid dysfunction, however, can also affect the interpretation of the HbA1c without affecting glucose levels. The lower erythrocyte turnover in overt hypothyroidism may result in higher HbA1c levels, which have been seen to decrease following the return to euthyroidism with minimal changes in glucose level [7,8].

The observations indicate that it is important to differentiate a genuine association between thyroid dysfunction and glycaemic status from changes in HbA1c due to non-glycaemic causes. The present study, therefore, assessed the prevalence and pattern of biochemical thyroid dysfunction in T2DM patients and its correlation with HbA1c, fasting glucose and postprandial glucose. The analysis also examined if there was any association that remained after the researchers adjusted for relevant demographic, clinical, and treatment-related factors.

Methods

Study design and eligibility criteria

The analysis was conducted in a retrospective cross sectional design and 350 records (one index encounter per record) were included. The study setting was specified as S.R. Patil Medical College, Badagandi, Bagalkote, Karnataka, India and the index encounter period was January 2026 to April 2026. Eligible encounters included clinically stable inpatients and adults (age ≥18 years) with known diabetes mellitus type 2 who were seen in general medicine or diabetology outpatient clinics. The values of thyroid-stimulating hormone (TSH), free thyroxine (free T4), and glycated haemoglobin (HbA1c) were needed within 14 days of the index encounter, and there were no major changes in treatment in the interim. Exclusion criteria were pregnancy, type 1 diabetes or gestational diabetes, acute metabolic decompensation or critical illness, conditions that affect the interpretation of the thyroid tests, and the lack of a valid index encounter that would allow for HbA1c reliability.

Thyroid status and glycaemic measures

The main exposure was current biochemical thyroid dysfunction and the main outcome was HbA1c (%). Reference intervals were 0.40–4.50 mIU/L for TSH, 0.80–1.80 ng/dL for free T4, and 2.30–4.20 pg/mL for free triiodothyronine (free T3). Normal TSH and free T4 were necessary for euthyroidism. Subclinical or overt hypothyroidism was defined as elevated TSH with normal or low levels of free T4, respectively. Subclinical hyperthyroidism was characterized by low TSH and normal free T4 and free T3. Overt hyperthyroidism was defined as suppressed TSH and elevated free T4 or free T3. If both TSH and free T4 were low, but free T3 was not available, it was considered indeterminate. The biochemical status and thyroid disease were recorded separately. Secondary outcomes were HbA1c ≥7%, fasting glucose and postprandial glucose.

Statistical analysis

Data for continuous variables were presented as mean (SD) or median (IQR) and for categorical variables as n (%). The prevalence of thyroid dysfunction was estimated for classifiable records, using a Wilson 95% CI. Welch's t-tests and Kruskal–Wallis tests were used to compare the thyroid dysfunction groups and the thyroid groups with ≥10 observations per group, respectively, with respect to the glycaemic measures. Spearman correlations with 95% bootstrap CIs (2,000 resamples) were used to evaluate the associations of TSH and free T4 with glycaemic measures.

The adjusted HbA1c differences were estimated using linear regression with HC3 robust standard errors. Crude and adjusted odds ratios for HbA1c ≥7% were estimated using logistic regression and the adjusted prevalence ratio was estimated using robust Poisson regression. Models adjusted for age, sex, diabetes duration, body mass index, insulin and metformin use, haemoglobin, and chronic kidney disease. Sensitivity analyses included modifications of eligibility, definition of thyroid exposure, adjustment for adherence, and glycaemic outcomes. Available observations were used in analyses without imputation. Indeterminate records were not included in the binary thyroid-status analyses.

Results

Cohort and thyroid status

The cohort included 350 records. Thyroid status was classifiable in 349 (99.7%). Of these, 89 (25.5%) had biochemical thyroid dysfunction (95% CI 21.2%–30.3%), most commonly subclinical hypothyroidism (Tables 1 and 2).

Thyroid status and glycaemic measures

Mean HbA1c was similar in the dysfunction and euthyroid groups (Figure 1). The unadjusted mean difference was 0.07 percentage points (95% CI −0.22 to 0.35). An exploratory comparison detected an overall difference in HbA1c across the four thyroid categories tested (p = 0.040). Free T4

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

showed a weak positive correlation with postprandial glucose (Spearman ρ = 0.164, p = 0.004). Neither TSH nor free T4 was significantly correlated with HbA1c (Tables 2 and 3).

Adjusted and sensitivity analyses

After adjustment, thyroid dysfunction was associated with an estimated HbA1c difference of 0.03 percentage points (95% CI −0.25 to 0.30). Estimates for HbA1c ≥7% and for the sensitivity analyses also had confidence intervals that included the null (Table 4 and Figure 2).

Table 1. Cohort characteristics

Characteristic	Overall (n = 350)
Age, years	55.6 (12.5)
Female sex	181 (51.7%)
Outpatient encounter	283 (80.9%)
Body mass index, kg/m²	25.4 (4.0)
Diabetes duration, years	6.9 (4.2–11.1)
Dyslipidaemia	182 (52.0%)
Hypertension	157 (44.9%)
Chronic kidney disease	25 (7.1%)
Metformin use	307 (87.7%)
Insulin use	81 (23.1%)
Prior thyroid disease	30 (8.6%)

Values are mean (SD), median (IQR), or n (%).

Table 2. Thyroid categories and glycaemic measures

Thyroid category	n (%)	HbA1c (%)	Fasting glucose (mg/dL)	Postprandial glucose (mg/dL)
Euthyroid	260 (74.3%)	260, 8.0 (7.2–8.6)	251, 149.0 (133.0–168.5)	235, 205.0 (177.0–232.0)
Subclinical hypothyroidism	57 (16.3%)	57, 8.3 (7.3–8.8)	54, 152.5 (129.0–168.5)	47, 197.0 (186.5–225.0)
Overt hypothyroidism	17 (4.9%)	17, 7.0 (6.8–7.8)	16, 149.5 (137.5–162.5)	14, 209.5 (171.8–217.0)
Subclinical hyperthyroidism	4 (1.1%)	4, 8.2 (7.9–8.6)	4, 131.5 (125.2–155.5)	4, 189.0 (176.5–219.0)
Overt hyperthyroidism	11 (3.1%)	11, 8.4 (7.9–8.7)	11, 150.0 (133.5–170.0)	10, 244.5 (185.5–265.2)
Indeterminate	1 (0.3%)	1, 6.5	1, 122.0	1, 168.0
Kruskal–Wallis H(3)	—	8.33	0.81	2.32
p	—	0.040	0.846	0.510

Glycaemic measures are shown as available n, median (IQR). Single values are shown for the indeterminate record. Subclinical hyperthyroidism and indeterminate status were excluded from the omnibus tests. The prevalence of dysfunction used the 349 classifiable records as its denominator.

Table 3. Glycaemic comparisons and hormone correlations

Measure	Euthyroid	Dysfunction	Difference (95% CI)	Welch t (df)	p
HbA1c, %	7.93 (1.11) n = 260	8.00 (1.18) n = 89	0.07 (−0.22 to 0.35)	0.46 (144.8)	0.646
Fasting glucose, mg/dL	150.39 (28.25) n = 251	151.80 (29.92) n = 85	1.41 (−5.92 to 8.73)	0.38 (138.1)	0.705
Postprandial glucose, mg/dL	204.49 (39.63) n = 235	205.33 (35.62) n = 75	0.84 (−8.77 to 10.45)	0.17 (137.3)	0.863

Group values are mean (SD). Differences are dysfunction minus euthyroid.

Measures	n	Spearman ρ	95% CI	p
TSH and HbA1c	350	−0.016	−0.118 to 0.087	0.762
TSH and Fasting glucose	337	0.006	−0.100 to 0.115	0.916
TSH and Postprandial glucose	311	−0.039	−0.149 to 0.073	0.496

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

Free T4 and HbA1c	350	0.072	−0.042 to 0.178	0.176
Free T4 and Fasting glucose	337	0.088	−0.021 to 0.192	0.106
Free T4 and Postprandial glucose	311	0.164	0.050 to 0.270	0.004

Correlation intervals are percentile bootstrap estimates. The correlation p-values are unadjusted for multiple comparisons.

Table 4. Regression and sensitivity analyses

Model or covariate	n	Estimate (95% CI)	Statistic	p
HbA1c model, adjusted β	—	—	—	—
Thyroid dysfunction	349	0.026 (−0.248 to 0.299)	t = 0.18	0.854
Age, per year	349	−0.001 (−0.010 to 0.009)	t = −0.15	0.883
Diabetes duration, per year	349	0.024 (0.004 to 0.045)	t = 2.32	0.021
Body mass index, per kg/m²	349	−0.017 (−0.046 to 0.012)	t = −1.12	0.263
Insulin use	349	0.514 (0.208 to 0.819)	t = 3.31	0.001
Metformin use	349	−0.097 (−0.458 to 0.263)	t = −0.53	0.595
Haemoglobin, per g/dL	349	0.077 (−0.024 to 0.178)	t = 1.49	0.137
Chronic kidney disease	349	0.020 (−0.609 to 0.648)	t = 0.06	0.951
Male sex	349	−0.257 (−0.527 to 0.014)	t = −1.87	0.063
HbA1c ≥7%, thyroid dysfunction	—	—	—	—
Crude OR	349	0.86 (0.48 to 1.55)	z = −0.50	0.619
Adjusted OR	349	0.82 (0.45 to 1.51)	z = −0.64	0.523
Adjusted PR	349	0.96 (0.85 to 1.09)	z = −0.58	0.563
Sensitivity analyses, adjusted β	—	—	—	—
No previously diagnosed thyroid disease	319	0.05 (−0.25 to 0.34)	t = 0.31	0.760
Hypothyroid spectrum vs euthyroid	334	−0.05 (−0.36 to 0.26)	t = −0.34	0.738
Haemoglobin ≥ 11 g/dL	339	−0.01 (−0.29 to 0.28)	t = −0.06	0.953
Additional adjustment for adherence	310	0.05 (−0.23 to 0.33)	t = 0.35	0.726
Fasting glucose as outcome	336	0.80 (−6.35 to 7.96)	t = 0.22	0.825
Postprandial glucose as outcome	310	−1.55 (−11.40 to 8.29)	t = −0.31	0.757

β denotes the adjusted mean difference in HbA1c percentage points, or mg/dL for the fasting and postprandial glucose sensitivity models. OR, odds ratio. PR, prevalence ratio estimated by robust Poisson regression. The adjusted models included age, sex, diabetes duration, body mass index, insulin use, metformin use, haemoglobin, and chronic kidney disease. The primary linear model used HC3 standard errors ($R^2 = 0.094$).

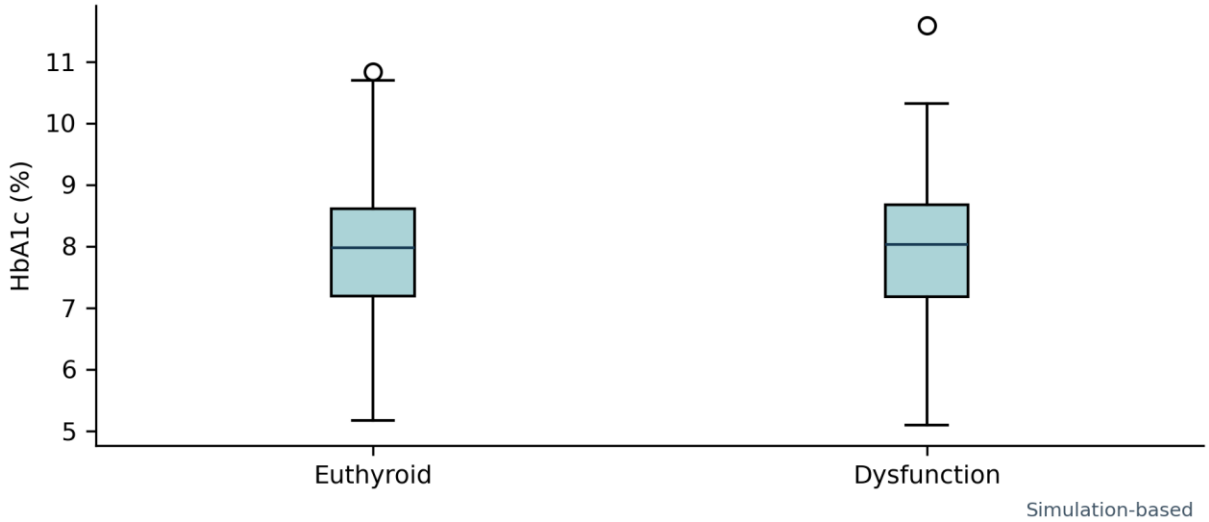

Figure 1. HbA1c by biochemical thyroid status. Boxes show the median and interquartile range for euthyroid (n = 260) and thyroid dysfunction (n = 89) records.

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

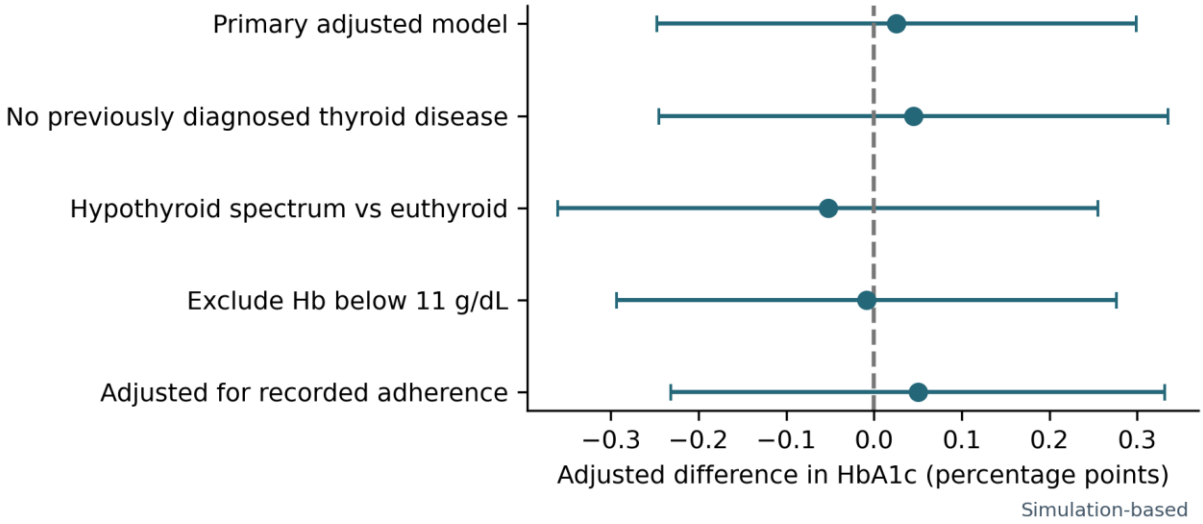

Figure 2. Adjusted difference in HbA1c by thyroid dysfunction status in the primary model and selected sensitivity analyses. Points represent model estimates and horizontal lines show 95% confidence intervals.

Discussion

In this study, biochemical thyroid dysfunction was found in 25.5% of the patients with T2DM, with the highest number being subclinical hypothyroidism. However, the prevalence of this condition is relatively high and glycaemic control was broadly similar between those with thyroid dysfunction and those who were euthyroid. The difference in mean HbA1c was not significant before adjustment, and after adjustment, the difference was 0.03 percentage points with the confidence interval spanning the null value. Thyroid dysfunction was also not linked to HbA1c ≥7% and there was no significant association between TSH or free T4 and HbA1c. The results indicate that biochemical thyroid dysfunction does not necessarily indicate worse glycaemic control in patients with both abnormalities.

The prevalence in this study is similar to some studies in diabetic people. Ozair et al. [9] conducted a study of 250 patients with T2DM from North India, and found thyroid dysfunction in 28% of the patients, with 18.8% being subclinical hypothyroidism. Patients with HbA1c ≥7%, dyslipidaemia, retinopathy and longer diabetes duration were more likely to have thyroid abnormalities. Their prevalence is very similar to our 25.5%, but the stronger association with poor glycaemic control is in contrast to our adjusted analysis, where thyroid status had little contribution to the variation in HbA1c.

In another study, Al-Geffari et al. [10] found thyroid dysfunction in 28.5% of 411 Saudi patients with T2DM, with hypothyroid disorders representing 25.3% and hyperthyroidism 3.2%. The risk factors were female sex and diabetes duration >10 years. Similar to our distribution, hypothyroid-spectrum abnormalities predominated, with diabetes duration being more strongly associated with HbA1c rather than an independent thyroid-associated glycaemic effect. This separation implies that the duration of diabetes may affect apparent associations between thyroid status and metabolic control.

There is a lack of consistency in the evidence on HbA1c. Cho et al. [11] studied 7,966 subjects and did not find a higher prevalence of subclinical hypothyroidism in T2DM compared to controls (7.4% versus 8.1%). However, in diabetic patients, HbA1c ≥9% was linked with higher odds of subclinical hypothyroidism than HbA1c <7% (OR 2.52), and this association was stronger in women (OR 4.58). Our study, however, did not show any relationship between thyroid dysfunction and dichotomous HbA1c ≥7% outcome, and continuous TSH was very little related to HbA1c.

This lack of a strong glycaemic relationship is confirmed by other studies. In the study by Sharma et al. [12] 205 patients with T2DM were studied and mean HbA1c levels were 8.33% in the patients with subclinical hypothyroidism and 7.89% in those who did not have subclinical hypothyroidism; however, the difference was not significant and TSH was not significantly associated with HbA1c. In a much larger group of 1,092 patients, Papazafiropoulou et al. [13] found thyroid dysfunction in 12.3% of patients, with no significant difference between those with and without thyroid dysfunction in HbA1c (7.38% vs. 7.81%). There was also a very similar duration of diabetes between groups. These results are very similar to our primary and sensitivity analyses.

The more recent Indian data are also informative. Rawat and Rawat [14] assessed 218 patients with T2DM and found that hypothyroidism was

prevalent in 58.7% of the patients, especially in women. Even with this prevalence, there was no significant difference in HbA1c levels between the euthyroid and hypothyroid groups, and there was no significant difference in major comorbidities. The absolute prevalence is significantly different from ours, perhaps due to classification differences and population differences, but the lack of meaningful separation in HbA1c is consistent with our results.

Other cohorts, however, have correlated thyroid dysfunction with more general diabetic complications despite the lack of direct glycaemic associations. In 500 South Indians, Reddy et al. [15] found thyroid dysfunction in 19.6% of whom 13.2% were subclinical hypothyroid. Subclinical hypothyroidism occurred more often in obese patients and diabetic retinopathy was significantly more prevalent in affected patients than in euthyroid patients (27.3% versus 8.9%). These observations indicate that thyroid status could be associated with metabolic phenotype or vascular risk in other ways that are not fully represented by HbA1c.

This weak positive correlation between free T4 and postprandial glucose (ρ=0.164) should, therefore, be interpreted with caution. The lack of association with HbA1c or fasting glucose, the small effect size, and multiple comparisons, are not supportive of a strong clinically relevant association. Similarly, the overall difference in HbA1c by thyroid category should not overpower the consistently null primary adjusted analysis. The multivariable model showed that diabetes duration and insulin use were more strongly associated with HbA1c than thyroid dysfunction, highlighting the potential for conventional measures of diabetes severity to be more important determinants of glycaemic control.

There are a number of restrictions to be taken into account. There were relatively few patients in some subgroups of the thyroid, which reduced the precision of some categories. Fasting and postprandial glucose were not recorded in all patients and biochemical thyroid classification might not be an accurate reflection of persistent thyroid disease if not repeated. Residual confounding is also a possibility. However, the robustness of the main finding after exclusion of previously diagnosed thyroid disease, restriction by haemoglobin concentration, further adjustment for adherence, and alternative glycaemic outcomes supports the finding that thyroid dysfunction was not independently associated with poorer glycaemic control in this population.

Conclusion

Thyroid dysfunction was prevalent in T2DM patients, and mostly represented by subclinical hypothyroidism. Thyroid dysfunction did not, however, have an independent association with HbA1c, poor glycaemic control, fasting glucose or postprandial glucose. The results of this study reinforce the notion that thyroid disease is a significant comorbidity in T2DM, and may not be associated with suboptimal glycaemic control.

References

1. Biondi B, Kahaly GJ, Robertson RP. Thyroid dysfunction and diabetes mellitus: two closely associated disorders. *Endocr Rev.* 2019;40(3):789–824. **doi:10.1210/er.2018-00163.**
2. Hadgu R, Worede A, Ambachew S. Prevalence of thyroid dysfunction and associated factors among adult type 2 diabetes mellitus patients, 2000–2022: a systematic review and meta-analysis. *Syst Rev.* 2024;13(1):119. **doi:10.1186/s13643-024-02527-y.**
3. Khassawneh AH, Al-Mistarehi AH, Zein Alaabdin AM, et al. Prevalence and predictors of thyroid dysfunction among type 2 diabetic patients: a case-control study. *Int J Gen Med.*2020;13:803–816. **doi:10.2147/IJGM.S273900.**
4. Jali MV, Kambar S, Jali SM, Pawar N, Nalawade P. Prevalence of thyroid dysfunction among type 2 diabetes mellitus patients. *Diabetes Metab Syndr.* 2017;11(Suppl 1)–S108. **doi:10.1016/j.dsx.2016.12.017.**
5. Elgazar EH, Esheba NE, Shalaby SA, Mohamed WF. Thyroid dysfunction prevalence and relation to glycemic control in patients with type 2 diabetes mellitus. *Diabetes Metab Syndr.* 2019;13(4):2513–2517. **doi:10.1016/j.dsx.2019.07.020.**
6. Sakyi SA, Ameyaw B, Laing EF, et al. Thyroid dysfunction and glycaemic control among type 2 diabetes mellitus patients in Ghana: a comparative cross-sectional study. *Endocrinol Diabetes Metab.* 2023;6(6). **doi:10.1002/edm2.447.**
7. Kim MK, Kwon HS, Baek KH, et al. Effects of thyroid hormone on A1C and glycated albumin levels in nondiabetic subjects with overt hypothyroidism. *Diabetes Care.* 2010;33(12):2546–2548. **doi:10.2337/dc10-0988.**
8. Bhattacharjee R, Thukral A, Chakraborty PP, et al. Effects of thyroid status on glycated hemoglobin. *Indian J Endocrinol Metab.* 2017;21(1):26–30. **doi:10.4103/2230-8210.196017.**
9. Ozair M, Noor S, Raghav A, Siddiqi SS, Chugtai AM, Ahmad J. Prevalence of thyroid disorders in North Indian Type 2 diabetic subjects: a cross-sectional study. *Diabetes Metab Syndr.* 2018;12(3):301–304. **doi:10.1016/j.dsx.2017.12.016.**
10. Al-Geffari M, Ahmad NA, Al-Sharqawi AH, Youssef AM, Alnaqeb D, Al-Rubeaan K. Risk factors for thyroid dysfunction among type 2 diabetic patients in a highly diabetes mellitus prevalent society. *Int J*

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.

Endocrinol.2013;2013:417920. **doi:10.1155/2013/417920.**
11. Cho JH, Kim HJ, Lee JH, et al. Poor glycemic control is associated with the risk of subclinical hypothyroidism in patients with type 2 diabetes mellitus. *Korean J Intern Med.* 2016;31(4):703–711. **doi:10.3904/kjim.2015.198.**
12. Sharma P, Sinha R, Prasad A, Mitra JK. Lack of association between poor glycemic control in T2DM and subclinical hypothyroidism. *J Thyroid Res.*2020;2020:8121395. **doi:10.1155/2020/8121395.**
13. Papazafiropoulou A, Sotiropoulos A, Kokolaki A, Kardara M, Stamataki P, Pappas S. Prevalence of thyroid dysfunction among Greek type 2 diabetic patients attending an outpatient clinic. *J Clin Med Res.* 2010;2(2):75–78. **doi:10.4021/jocmr2010.03.281w.**
14. Rawat J, Rawat A. Prevalence and impact of hypothyroidism on glycaemic control in Indian patients with type 2 diabetes mellitus: a cross-sectional study. *J ASEAN Fed Endocr Soc.* 2026;41(1):82–86. **doi:10.15605/jafes.41.1.4989.**
15. Reddy N, Pradeep TVS, Tirupati S, Sarathi V, Kumar D. Thyroid dysfunction and its association with microvascular complications in patients with type 2 diabetes mellitus in South India. *Diabetes Metab Syndr.* 2020;14(4):615–617. **doi:10.1016/j.dsx.2020.05.005.**

 © 2026 Dr. Shivaraj Hadimani et al. Open Access under the Creative Commons Attribution 4.0 International License.