

Prevalence, Patterns, and Factors Associated with Thyroid Dysfunction among Adult Patients with Type 2 Diabetes Mellitus Attending a Tertiary Care Hospital: A Retrospective Record-Based Study

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ABSTRACT

Background: Thyroid dysfunction frequently coexists with type 2 diabetes mellitus (T2DM) and may adversely affect glycaemic control, lipid metabolism and vascular outcomes. However, thyroid abnormalities, particularly subclinical disorders, may remain clinically unrecognised among patients with T2DM. **Objectives:** To determine the prevalence and patterns of thyroid dysfunction and identify associated demographic, clinical and biochemical factors among adult patients with T2DM attending a tertiary-care hospital. **Methods:** A hospital-based retrospective record review was conducted in the Department of General Medicine at BGS Nagarur, Bengaluru, from April to July 2026. Medical records of 277 adults with T2DM and available thyroid-function test results were reviewed. Thyroid status was classified using serum thyroid-stimulating hormone and free thyroxine values. Descriptive statistics, chi-square test, independent-samples *t*-test, Mann Whitney *U* test and binary logistic regression were applied. Adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were reported. **Results:** Thyroid dysfunction was identified in 58 patients, corresponding to a prevalence of 20.9% (95% CI: 16.6–26.1%). Subclinical hypothyroidism was the predominant pattern (11.9%), followed by overt hypothyroidism (5.8%), subclinical hyperthyroidism (1.8%) and overt hyperthyroidism (1.4%). Of the affected patients, 56.9% had newly identified biochemical thyroid dysfunction. Thyroid dysfunction was significantly associated with female sex, higher body mass index (BMI), poor glycaemic control, dyslipidaemia and diabetic retinopathy. After adjustment, female sex (AOR: 2.04; 95% CI: 1.07–3.88), BMI (AOR per kg/m²: 1.21; 95% CI: 1.10–1.33), diabetes duration (AOR per year: 1.07; 95% CI: 1.00–1.15) and HbA1c (AOR per 1% increase: 1.44; 95% CI: 1.12–1.85) remained independently associated with thyroid dysfunction. **Conclusion:** Thyroid dysfunction affected approximately one-fifth of patients with T2DM, with subclinical hypothyroidism being the most common pattern. Targeted thyroid-function assessment may be beneficial, particularly among women and patients with obesity, longer diabetes duration or poor glycaemic control.

Keywords: body mass index; glycaemic control; hypothyroidism; prevalence; thyroid dysfunction; type 2 diabetes mellitus.

INTRODUCTION

Diabetes mellitus and thyroid dysfunction are among the most frequently encountered endocrine disorders, and their coexistence may adversely influence metabolic control and the development of vascular complications. Thyroid hormones regulate carbohydrate metabolism, intestinal glucose absorption, hepatic gluconeogenesis, lipid metabolism and insulin sensitivity. Conversely, diabetes may alter

hypothalamic–pituitary–thyroid-axis activity, thyroid-hormone metabolism and peripheral conversion of thyroxine to triiodothyronine.¹ This bidirectional interaction can complicate the clinical management of patients with type 2 diabetes mellitus (T2DM), particularly because mild or subclinical thyroid dysfunction may remain clinically unrecognised.² The prevalence of thyroid dysfunction among patients with T2DM varies considerably according to age, sex,

ethnicity, iodine status, laboratory reference ranges, inclusion of previously diagnosed disease and the characteristics of the population studied. A recent systematic review and meta-analysis of 38 studies involving 19,803 adults with T2DM reported a pooled thyroid-dysfunction prevalence of 20.24%. Subclinical hypothyroidism was the most common abnormality, with a pooled prevalence of 11.87%, followed by overt hypothyroidism, subclinical hyperthyroidism and overt hyperthyroidism.³

Indian studies have similarly demonstrated an appreciable burden of thyroid abnormalities among patients with T2DM. Hypothyroidism, particularly its subclinical form, has been the predominant pattern, and female sex, obesity, longer diabetes duration and poor glycaemic control have been reported as potential associated factors.⁴ A study from rural South India also documented the coexistence of thyroid disorders and diabetes, highlighting the relevance of thyroid evaluation in Indian diabetes-care settings.⁵ Large multicentre investigations have further shown that the prevalence and pattern of thyroid dysfunction may vary with demographic characteristics, glycaemic status and duration of diabetes.⁶

The coexistence of these disorders is clinically important because unrecognised thyroid dysfunction may contribute to weight gain, dyslipidaemia, suboptimal glycaemic control and increased cardiovascular risk. However, evidence from retrospective tertiary-care settings in Bengaluru remains limited. Therefore, the present study was conducted to determine the prevalence and patterns of thyroid dysfunction among adult patients with T2DM attending a tertiary-care hospital and to evaluate the demographic, clinical and biochemical factors associated with thyroid dysfunction.

MATERIALS AND METHODS

A hospital-based retrospective record review was conducted in the Department of General Medicine at BGS Nagarur, Bengaluru, between April and July 2026. Medical records of adult patients with type 2 diabetes mellitus (T2DM) who attended the inpatient or outpatient services during the study period were reviewed.

Patients aged ≥ 18 years with a documented diagnosis of T2DM and available thyroid-function test results were included. Records contained serum thyroid-stimulating hormone (TSH) and free thyroxine (FT4) values sufficient to classify thyroid status.

Patients with type 1 diabetes, gestational diabetes, secondary diabetes, pregnancy, postpartum status, critical illness or euthyroid sick syndrome were excluded. Patients receiving amiodarone, lithium or high-dose glucocorticoids were also excluded. Records with incomplete thyroid-function results, insufficient clinical information or duplicate entries were not considered.

The sample size was estimated using the single-population proportion formula:

$$n = \frac{Z_{\alpha/2}^2 p(1-p)}{d^2}$$

A pooled thyroid-dysfunction prevalence of 20.24% among patients with T2DM was obtained from a systematic review and meta-analysis by Hadgu et al.³ At a 95% confidence level, 5% absolute precision and an anticipated prevalence of 20.24%, the minimum calculated sample size was 249. After allowing for 10% incomplete or unusable records, the final required sample size was 277.

Eligible records were selected consecutively in chronological order until the required sample size was reached. Each patient was included only once. When multiple thyroid-function tests were available, the result closest to the index diabetes visit was considered.

Data were retrieved from medical records, the hospital information system and laboratory reports using a structured data-extraction form. The variables collected included age, sex, body mass index (BMI), duration of diabetes, treatment modality, hypertension, dyslipidaemia and documented diabetic complications. Laboratory variables included fasting blood glucose, postprandial blood glucose, glycated haemoglobin (HbA1c), lipid profile, serum creatinine, TSH, FT4 and free triiodothyronine, where available.

The index date was defined as the date of thyroid-function testing. HbA1c and other biochemical measurements obtained within three months before or after the index date were included. When multiple values were available, the value closest to the index date was used.

Operational definitions

T2DM was identified from a physician-documented diagnosis or ongoing treatment for T2DM. Thyroid status was classified according to the reference intervals used by the institutional laboratory.

Euthyroidism was defined as TSH and FT4 values within their respective reference ranges. Subclinical hypothyroidism was defined as elevated TSH with normal FT4, whereas overt hypothyroidism was defined as elevated TSH with reduced FT4. Subclinical hyperthyroidism was defined as reduced TSH with normal FT4 and FT3, while overt hyperthyroidism was defined as reduced TSH with elevated FT4 and/or FT3. Previously diagnosed thyroid dysfunction was identified from a documented thyroid diagnosis or current use of thyroid-specific medication. Newly identified thyroid dysfunction was defined as an abnormal thyroid-function profile in a patient without previously documented thyroid disease.

Glycaemic control was classified as adequate when HbA1c was $< 7\%$ and poor when HbA1c was $\geq 7\%$. BMI was calculated as weight in kilograms divided by

height in metres squared. Duration of diabetes was calculated from the recorded date of diagnosis to the index date.

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 22. Categorical variables were summarised as frequencies and percentages. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation, while skewed variables were reported as median and interquartile range.

Ethical approval was obtained from the Institutional Ethics Committee. A waiver of informed consent was obtained because the study involved retrospective review of existing records. Patient identifiers were removed, and confidentiality was maintained throughout the study.

The prevalence of thyroid dysfunction was presented with a 95% confidence interval. Categorical variables were compared using Pearson's chi-square test or Fisher's exact test. Continuous variables were compared using the independent-samples *t*-test or Mann-Whitney *U*-test, as appropriate.

Binary logistic regression was performed to identify factors independently associated with thyroid dysfunction. Clinically relevant variables and those with $p < 0.20$ in the univariable analysis were considered for multivariable analysis. Adjusted odds ratios with 95% confidence intervals were reported. A two-sided $p < 0.05$ was considered statistically significant.

RESULTS

A total of 277 patients with type 2 diabetes mellitus were included in the study. Their mean age was 54.62 ± 9.87 years; 183 (66.1%) were aged 40–59 years and 85 (30.7%) were aged ≥ 60 years. The sex distribution was nearly equal, with 138 (49.8%) males and 139 (50.2%) females. The mean body mass index was 26.53 ± 3.56 kg/m²; 146 (52.7%) participants were overweight and 44 (15.9%) were obese. The median duration of diabetes was 6.02 years (IQR: 3.70–9.47), and 62 (22.4%) participants had diabetes for more than 10 years. The mean HbA1c was $7.91 \pm 1.28\%$, with 208 (75.1%) participants demonstrating poor glycaemic control (HbA1c $\geq 7\%$). Oral glucose-lowering medication alone was used by 150 (54.2%) participants, while 95 (34.3%) received a combination of oral medication and insulin. Dyslipidaemia and hypertension were present in 115 (41.5%) and 93 (33.6%) participants, respectively. Neuropathy, retinopathy and nephropathy were documented in 16.2%, 15.9% and 15.2% of participants, respectively (Table 1).

Of the 277 patients evaluated, 58 had thyroid dysfunction, giving an overall prevalence of 20.9% (95% CI: 16.6–26.1%), while 219 (79.1%) were euthyroid. Subclinical hypothyroidism was the most frequently observed thyroid disorder, affecting 33 (11.9%) participants. Overt hypothyroidism was

identified in 16 (5.8%) participants, whereas subclinical and overt hyperthyroidism were observed in five (1.8%) and four (1.4%) participants, respectively (Table 2).

Among the 58 patients with thyroid dysfunction, 25 (43.1%; 95% CI: 31.2–55.9%) had a previously documented thyroid disorder, whereas 33 (56.9%; 95% CI: 44.1–68.8%) had newly identified biochemical thyroid dysfunction. Thus, more than half of the thyroid dysfunction detected in the study population had not been previously diagnosed (Table 2).

The prevalence of thyroid dysfunction did not differ significantly across age groups ($\chi^2 = 0.927$, $p = 0.629$). However, thyroid dysfunction was significantly more frequent among females than males (27.3% versus 14.5%; $\chi^2 = 6.148$, $p = 0.013$). A significant association was also observed between BMI category and thyroid dysfunction ($\chi^2 = 19.499$, $p < 0.001$). The prevalence increased from 6.9% among underweight or normal-weight participants to 24.0% among overweight participants and 38.6% among participants with obesity (Table 3).

The prevalence of thyroid dysfunction increased from 16.2% among patients with a diabetes duration of ≤ 5 years to 25.8% among those with a duration exceeding 10 years; however, the categorical association was not statistically significant ($\chi^2 = 2.670$, $p = 0.263$). Thyroid dysfunction was significantly more common among patients with poor glycaemic control than among those with HbA1c $< 7\%$ (24.0% versus 11.6%; $\chi^2 = 4.124$, $p = 0.042$). Patients with dyslipidaemia also had a significantly higher prevalence of thyroid dysfunction than those without dyslipidaemia (27.8% versus 16.0%; $\chi^2 = 4.946$, $p = 0.026$). The association with hypertension approached but did not reach statistical significance (28.0% versus 17.4%; $p = 0.059$) (Table 3).

Thyroid dysfunction was present in 45.5% of patients with diabetic retinopathy compared with 16.3% of those without retinopathy, and this association was statistically significant ($\chi^2 = 17.272$, $p < 0.001$). Although thyroid dysfunction was more frequent among patients with nephropathy (28.6%) and neuropathy (28.9%) than among those without these complications (19.6% and 19.4%, respectively), neither association reached statistical significance ($p = 0.265$ and $p = 0.218$, respectively) (Table 3).

Patients with thyroid dysfunction had a significantly higher mean BMI than euthyroid patients (28.28 ± 3.18 versus 26.07 ± 3.52 kg/m²; $p < 0.001$). They also had a longer median duration of diabetes—7.56 years (IQR: 4.38–10.61) compared with 5.74 years (IQR: 3.47–8.79) among euthyroid patients ($p = 0.026$). The mean age was slightly higher in the thyroid-dysfunction group, but the difference was not statistically significant (56.34 ± 10.50 versus 54.16 ± 9.67 years; $p = 0.158$) (Table 4).

Patients with thyroid dysfunction had significantly higher fasting blood glucose levels than euthyroid

patients (168.0 ± 48.0 versus 154.0 ± 44.0 mg/dL; $p = 0.048$). Similarly, their mean HbA1c was significantly higher ($8.39 \pm 1.12\%$ versus $7.79 \pm 1.30\%$; $p = 0.001$). Postprandial blood glucose was also higher in the thyroid-dysfunction group, although the difference did not reach statistical significance (244.0 ± 71.0 versus 225.0 ± 66.0 mg/dL; $p = 0.069$) (Table 4).

The thyroid-dysfunction group had higher mean total cholesterol, triglyceride and LDL-cholesterol levels and a lower mean HDL-cholesterol level than the euthyroid group. However, none of these lipid parameters differed significantly between the groups. Serum creatinine was also comparable between patients with thyroid dysfunction and euthyroid patients (1.05 ± 0.42 versus 0.98 ± 0.36 mg/dL; $p = 0.249$) (Table 4).

In the univariable logistic regression analysis, female sex was associated with more than twice the odds of thyroid dysfunction compared with male sex (crude OR: 2.22; 95% CI: 1.20–4.11; $p = 0.011$). Increasing BMI, longer duration of diabetes and higher HbA1c

were also significantly associated with thyroid dysfunction. Age, analysed per 10-year increase, was not significantly associated with thyroid dysfunction in the unadjusted analysis (crude OR: 1.24; 95% CI: 0.91–1.69; $p = 0.158$) (Table 5).

After simultaneous adjustment for age, sex, BMI, duration of diabetes and HbA1c, female sex remained independently associated with thyroid dysfunction (adjusted OR: 2.04; 95% CI: 1.07–3.88; $p = 0.029$). Each 1-kg/m² increase in BMI was associated with a 21% increase in the odds of thyroid dysfunction (adjusted OR: 1.21; 95% CI: 1.10–1.33; $p < 0.001$). Each additional year of diabetes was associated with a 7% increase in the odds of thyroid dysfunction (adjusted OR: 1.07; 95% CI: 1.00–1.15; $p = 0.043$), while each 1% increase in HbA1c was associated with a 44% increase in the odds (adjusted OR: 1.44; 95% CI: 1.12–1.85; $p = 0.005$). Age was not independently associated with thyroid dysfunction after adjustment (adjusted OR per 10-year increase: 1.34; 95% CI: 0.97–1.85; $p = 0.075$) (Table 5).

Table 1. Baseline demographic and clinical characteristics of the study participants

Characteristic	Total (N = 277)
Age, years, mean $\pm$ SD	54.62 $\pm$ 9.87
Age group, n (%)	
18–39 years	9 (3.2)
40–59 years	183 (66.1)
≥ 60 years	85 (30.7)
Sex, n (%)	
Male	138 (49.8)
Female	139 (50.2)
BMI, kg/m ² , mean $\pm$ SD	26.53 $\pm$ 3.56
BMI category, n (%)	
Underweight or normal weight	87 (31.4)
Overweight	146 (52.7)
Obese	44 (15.9)
Duration of diabetes, years, median (IQR)	6.02 (3.70–9.47)
Duration of diabetes, n (%)	
≤ 5 years	111 (40.1)
6–10 years	104 (37.5)
> 10 years	62 (22.4)
HbA1c, %, mean $\pm$ SD	7.91 $\pm$ 1.28
Glycaemic-control status, n (%)	
HbA1c $< 7\%$	69 (24.9)
HbA1c $\geq 7\%$	208 (75.1)
Diabetes treatment, n (%)	
Oral glucose-lowering medication alone	150 (54.2)

Insulin alone	32 (11.6)
Oral medication plus insulin	95 (34.3)
Comorbidities, n (%)	
Hypertension	93 (33.6)
Dyslipidaemia	115 (41.5)
Diabetic complications, n (%)	
Retinopathy	44 (15.9)
Nephropathy	42 (15.2)
Neuropathy	45 (16.2)

BMI: body mass index; HbA1c: glycated haemoglobin; IQR: interquartile range; SD: standard deviation.

Table 2. Prevalence and patterns of thyroid dysfunction among patients with type 2 diabetes mellitus

Thyroid status	Frequency, n	Prevalence, %	95% CI
Euthyroid	219	79.1	73.9–83.4
Any thyroid dysfunction	58	20.9	16.6–26.1
Subclinical hypothyroidism	33	11.9	8.6–16.3
Overt hypothyroidism	16	5.8	3.6–9.2
Subclinical hyperthyroidism	5	1.8	0.8–4.2
Overt hyperthyroidism	4	1.4	0.6–3.7

Table 3. Diagnostic status among patients with thyroid dysfunction

Diagnostic status	Frequency, n	Percentage ¹	95% CI
Previously diagnosed thyroid dysfunction	25	43.1	31.2–55.9
Newly identified biochemical thyroid dysfunction	33	56.9	44.1–68.8

¹The denominator was 58 patients with thyroid dysfunction.

Table 3. Association of demographic and clinical characteristics with thyroid dysfunction

Characteristic		Total, n	Thyroid dysfunction, n (%) ¹	Euthyroid, n (%) ¹	χ^2 value	p-value
Age group	18–39 years	9	1 (11.1)	8 (88.9)	0.927	0.629
	40–59 years	183	37 (20.2)	146 (79.8)		
	≥60 years	85	20 (23.5)	65 (76.5)		
Sex	Male	138	20 (14.5)	118 (85.5)	6.148	0.013*
	Female	139	38 (27.3)	101 (72.7)		
BMI category	Underweight/normal	87	6 (6.9)	81 (93.1)	19.5	<0.001*
	Overweight	146	35 (24.0)	111 (76.0)		
	Obese	44	17 (38.6)	27 (61.4)		
Duration of diabetes	≤5 years	111	18 (16.2)	93 (83.8)	2.67	0.263
	6–10 years	104	24 (23.1)	80 (76.9)		
	>10 years	62	16 (25.8)	46 (74.2)		
Glycaemic control	HbA1c <7%	69	8 (11.6)	61 (88.4)	4.124	0.042*
	HbA1c ≥7%	208	50 (24.0)	158 (76.0)		
Hypertension	Absent	184	32 (17.4)	152 (82.6)	3.552	0.059
	Present	93	26 (28.0)	67 (72.0)		

Dyslipidaemia	Absent	162	26 (16.0)	136 (84.0)	4.946	0.026*
	Present	115	32 (27.8)	83 (72.2)		
Diabetic retinopathy	Absent	233	38 (16.3)	195 (83.7)	17.27	<0.001*
	Present	44	20 (45.5)	24 (54.5)		
Diabetic nephropathy	Absent	235	46 (19.6)	189 (80.4)	1.241	0.265
	Present	42	12 (28.6)	30 (71.4)		
Diabetic neuropathy	Absent	232	45 (19.4)	187 (80.6)	1.518	0.218
	Present	45	13 (28.9)	32 (71.1)		

¹Row percentages were calculated.

Pearson's chi-square test was used.

BMI: body mass index; HbA1c: glycated haemoglobin.

Table 4. Comparison of clinical and biochemical parameters according to thyroid status

Parameter	Thyroid dysfunction (n = 58)	Euthyroid (n = 219)	p-value
Age, years	56.34 ± 10.50	54.16 ± 9.67	0.158
BMI, kg/m ²	28.28 ± 3.18	26.07 ± 3.52	<0.001*
Duration of diabetes, years ¹	7.56 (4.38–10.61)	5.74 (3.47–8.79)	0.026*#
Fasting blood glucose, mg/dL	168.0 ± 48.0	154.0 ± 44.0	0.048*
Postprandial blood glucose, mg/dL	244.0 ± 71.0	225.0 ± 66.0	0.069
HbA1c, %	8.39 ± 1.12	7.79 ± 1.30	0.001*
Total cholesterol, mg/dL	189.0 ± 42.0	181.0 ± 39.0	0.194
Triglycerides, mg/dL	182.0 ± 78.0	166.0 ± 70.0	0.160
LDL cholesterol, mg/dL	112.0 ± 35.0	106.0 ± 32.0	0.241
HDL cholesterol, mg/dL	42.0 ± 9.0	44.0 ± 10.0	0.145
Serum creatinine, mg/dL	1.05 ± 0.42	0.98 ± 0.36	0.249

Independent-samples *t*-test.

Mann–Whitney *U* test.

BMI: body mass index; HbA1c: glycated haemoglobin; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

Table 5. Factors associated with thyroid dysfunction: univariable and multivariable logistic regression

Predictor	Unit/comparison	Crude OR (95% CI) ¹	Unadjusted p-value	Adjusted OR (95% CI)	Adjusted p-value
Age	Per 10-year increase	1.24 (0.91–1.69)	0.158	1.34 (0.97–1.85)	0.075
Female sex	Female vs male	2.22 (1.20–4.11)	0.011*	2.04 (1.07–3.88)	0.029*
BMI	Per 1-kg/m ² increase	1.20 (1.10–1.31)	<0.001*	1.21 (1.10–1.33)	<0.001*
Duration of diabetes	Per one-year increase	1.08 (1.02–1.14)	0.012*	1.07 (1.00–1.15)	0.043*
HbA1c	Per 1% increase	1.47 (1.17–1.86)	0.001*	1.44 (1.12–1.85)	0.005*

DISCUSSION

The prevalence of thyroid dysfunction among patients with T2DM in the present study was 20.9%, indicating that approximately one in five patients was affected. This finding closely corresponded with the pooled prevalence of 20.24% reported by Hadgu et al.³ Differences in prevalence across studies may be

attributed to variations in age, sex, iodine status, laboratory reference ranges and inclusion of previously diagnosed thyroid disease.

Subclinical hypothyroidism was the most common abnormality, affecting 11.9% of participants, followed by overt hypothyroidism in 5.8%. Subclinical and overt hyperthyroidism were relatively uncommon. This

pattern was consistent with the meta-analysis by Hadgu et al., which reported subclinical hypothyroidism as the predominant thyroid abnormality among patients with T2DM.³ Similar findings have also been reported in Indian studies.^{4,5} More than half of the affected participants had newly identified biochemical thyroid dysfunction, suggesting that a substantial proportion of thyroid abnormalities may remain clinically unrecognised.

Thyroid dysfunction was significantly more frequent among women, and female sex remained independently associated with approximately twice the odds of thyroid dysfunction. Comparable findings were reported by Khassawneh et al. and Ogbonna and Ezeani.^{7,8} The greater susceptibility among women may be related to autoimmune predisposition, sex-hormone influences and X-chromosome-related immune mechanisms.

Increasing BMI was independently associated with thyroid dysfunction. Patients with obesity had a higher prevalence than overweight or normal-weight patients, and each 1-kg/m² increase in BMI was associated with a 21% increase in adjusted odds. Obesity and thyroid dysfunction may have a bidirectional relationship, as hypothyroidism can promote weight gain, while obesity and insulin resistance may influence TSH secretion and thyroid-hormone metabolism.^{1,2}

Patients with thyroid dysfunction had significantly higher fasting blood glucose and HbA1c levels. Poor glycaemic control was associated with a higher prevalence of thyroid dysfunction, and each 1% increase in HbA1c was associated with a 44% increase in adjusted odds. Sakyi et al. similarly reported an association between thyroid dysfunction and glycaemic control.⁹ However, Iwakura et al. found that baseline thyroid-function indices did not predict subsequent glycaemic control.¹⁰ Therefore, the present association should not be interpreted as causal.

Longer diabetes duration was also independently associated with thyroid dysfunction, with a 7% increase in adjusted odds for each additional year of diabetes. Chronic insulin resistance, inflammation and cumulative metabolic disturbances may contribute to this relationship. Longitudinal studies have also demonstrated that thyroid dysfunction may develop during follow-up among patients with diabetes.^{11,12}

Dyslipidaemia and diabetic retinopathy showed significant unadjusted associations with thyroid dysfunction. Nevertheless, individual lipid parameters were not significantly different between thyroid-dysfunction and euthyroid groups. The observed association with retinopathy may reflect longer diabetes duration, poor glycaemic control or shared vascular risk factors. Nephropathy and neuropathy were not significantly associated with thyroid dysfunction.

These findings support thyroid-function assessment among patients with T2DM, particularly women and patients with obesity, longer diabetes duration or

inadequate glycaemic control. However, the retrospective, single-centre design, possible selection of patients more likely to undergo thyroid testing, incomplete availability of thyroid-antibody results and inability to establish temporality should be considered. Prospective multicentre studies are needed to determine whether targeted thyroid screening improves metabolic and clinical outcomes.

Strengths

This study has several strengths. First, it included a well-defined cohort of 277 adult patients with type 2 diabetes mellitus and used clearly specified biochemical criteria to classify different patterns of thyroid dysfunction. Second, the study evaluated not only the prevalence and pattern of thyroid dysfunction but also a broad range of demographic, clinical and biochemical factors, including BMI, diabetes duration, glycaemic control, dyslipidaemia and diabetic complications. Third, the use of multivariable logistic regression allowed independent associations to be assessed after adjustment for clinically relevant factors. Fourth, the study distinguished previously diagnosed thyroid disease from newly identified biochemical thyroid dysfunction, providing clinically relevant information regarding potentially unrecognised thyroid abnormalities. Finally, the study provides data from a tertiary-care setting in Bengaluru and adds to the limited Indian evidence on thyroid dysfunction among patients with type 2 diabetes mellitus.

Limitations

This study has several limitations. First, its retrospective, single-centre design limits the generalisability of the findings to other healthcare settings and populations. Second, because thyroid-function testing was based on clinical records, selection bias is possible, as patients who underwent thyroid testing may differ from those who were not tested. Third, the study relied on routinely recorded clinical and laboratory data, and some potentially relevant variables, including thyroid autoantibody status, dietary iodine intake, family history of thyroid disease, medication adherence and detailed socioeconomic characteristics, were not consistently available. Fourth, the cross-sectional assessment of thyroid function and associated factors does not allow temporal or causal relationships to be established. Fifth, thyroid-function classification was based on the reference intervals of the institutional laboratory, which may limit direct comparison with studies using different assay methods or reference ranges. Finally, residual confounding cannot be excluded despite multivariable adjustment. Larger prospective, multicentre studies with standardised thyroid assessment and longer follow-up are warranted to confirm these findings.

CONCLUSION

Thyroid dysfunction was observed in approximately one-fifth of patients with type 2 diabetes mellitus, with subclinical hypothyroidism being the predominant pattern. A substantial proportion of cases were newly identified, indicating that thyroid abnormalities may remain clinically unrecognised in this population. Female sex, higher body mass index, longer duration of diabetes and poor glycaemic control were independently associated with thyroid dysfunction. These findings support targeted thyroid-function assessment, particularly among high-risk patients with type 2 diabetes. Early recognition and appropriate management of coexisting thyroid dysfunction may improve metabolic management and reduce related complications. Prospective multicentre studies are required to confirm these findings.

Declarations

Ethics Approval and Consent to Participate

The study was conducted in accordance with the ethical standards applicable to retrospective research involving human participants. Ethical approval was obtained from the Institutional Ethics Committee. A waiver of informed consent was granted because the study involved retrospective review of existing medical records and involved no direct participant intervention. Patient identifiers were removed, and confidentiality was maintained throughout the study.

Consent for Publication

Not applicable. The study used retrospectively collected, de-identified medical record data, and no individually identifiable information is reported.

Availability of Data and Materials

The datasets generated and/or analysed during the current study are not publicly available because they contain information derived from patient medical records. De-identified data may be made available from the corresponding author on reasonable request, subject to institutional ethical and data-protection requirements.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

All authors contributed to the conception and design of the study, acquisition and interpretation of data, drafting or critical revision of the manuscript, and approved the final version of the manuscript.

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Use of Generative Artificial Intelligence

No generative artificial intelligence tools were used in the conception, data collection, statistical analysis, interpretation of results, or preparation of the manuscript. The authors take full responsibility for the content of the manuscript.

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