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Thyroid Function Profile in Adults with Type 2 Diabetes Mellitus Compared With Healthy Controls: A Hospital-Based Case-Control Study.

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Abstract

Background: Type 2 diabetes mellitus and thyroid dysfunction frequently coexist and can interact through changes in insulin sensitivity, hepatic glucose production, lipid metabolism, and peripheral thyroid hormone conversion. Unrecognized thyroid abnormalities can complicate metabolic control and clinical assessment in adults with diabetes. ***Objectives:*** To compare serum triiodothyronine, thyroxine, thyroid-stimulating hormone, and fasting plasma glucose concentrations between adults with type 2 diabetes mellitus and healthy controls. ***Methods:*** This hospital-based case-control study included 50 adults aged 20-70 years, comprising 25 patients with type 2 diabetes mellitus and 25 apparently healthy controls. Participants with known thyroid disease, hepatobiliary disease, pregnancy, or use of medicines affecting thyroid function were excluded. Fasting plasma glucose was measured by the glucose oxidase-peroxidase method, while thyroid hormones were estimated using chemiluminescence immunoassay. Continuous variables were summarized as mean and standard deviation and compared using an independent-samples t test. ***Results:*** The mean age was 39.76 ± 9.39 years in the diabetes group and 34.36 ± 12.06 years in the control group. Mean fasting plasma glucose was significantly higher in patients with diabetes than in controls (158.00 ± 50.13 versus 94.16 ± 8.87 mg/dL). Patients with diabetes had lower mean triiodothyronine (0.53 ± 0.47 versus 1.22 ± 0.38 ng/mL) and thyroxine (48.47 ± 21.75 versus 95.25 ± 19.38 nmol/L), together with higher thyroid-stimulating hormone (23.83 ± 12.60 versus 1.95 ± 0.78 mIU/L). All biochemical differences were statistically significant. ***Conclusion:*** Adults with type 2 diabetes demonstrated a biochemical pattern consistent with reduced thyroid function. Thyroid assessment is clinically relevant in diabetic patients, particularly when glycaemic control is poor or symptoms suggest thyroid dysfunction.

Keywords: type 2 diabetes mellitus; thyroid dysfunction; hypothyroidism; thyroid-stimulating hormone; triiodothyronine; thyroxine.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major global metabolic disorder characterized by insulin resistance, progressive beta-cell dysfunction, and persistent hyperglycaemia. The International Diabetes Federation estimated that 537 million adults were living with diabetes in 2021 and projected a substantial increase by 2045.[1] The expanding burden is especially relevant to low- and middle-income countries, where delayed diagnosis and coexisting endocrine disorders increase the complexity of long-term care. Beyond vascular complications, T2DM is associated with disturbances in several hormonal axes, including thyroid hormone regulation.

Diabetes and thyroid disease have a bidirectional relationship. Thyroid hormones influence glucose absorption, hepatic gluconeogenesis, insulin secretion, lipid turnover, resting energy expenditure, and peripheral glucose utilization. Conversely, hyperglycaemia, insulin resistance, obesity, systemic inflammation, and diabetes-related illness can alter hypothalamic-pituitary-thyroid signalling and the peripheral conversion of thyroxine (T4) to triiodothyronine (T3).[2,3] These interactions create a spectrum ranging from subclinical hypothyroidism to overt thyroid dysfunction or a low-T3 pattern. The clinical manifestations can overlap with those of diabetes, including fatigue, weight change, dyslipidaemia, neuropathic symptoms, and cardiovascular complaints, which can delay recognition.

Thyroid dysfunction is common in the general population, although prevalence varies with age, sex, iodine status, autoimmunity, and the diagnostic thresholds applied.[4] Studies in diabetic populations consistently report higher frequencies than those observed in unselected adults. Perros and colleagues documented the value of periodic thyroid assessment in patients with diabetes, while subsequent evidence has emphasized the predominance of hypothyroid states.[5] A meta-analysis found a significant association between subclinical hypothyroidism and T2DM, and a recent systematic review

estimated that approximately one fifth of adults with T2DM had some form of thyroid dysfunction.[6,7] Indian data have similarly demonstrated clinically relevant thyroid abnormalities among patients attending diabetes services. Identifying thyroid dysfunction in T2DM has practical importance because untreated hypothyroidism can aggravate dyslipidaemia, weight gain, endothelial dysfunction, and cardiovascular risk, while hyperthyroidism can worsen hyperglycaemia and catabolism. In addition, altered thyroid hormone concentrations can reflect poor metabolic control, concurrent illness, medication exposure, or genuine primary thyroid disease. Local laboratory comparisons are useful for defining the magnitude and direction of these abnormalities in the population served by a hospital. The primary objective of this study was to compare serum T3, T4, thyroid-stimulating hormone (TSH), and fasting plasma glucose concentrations between adults with T2DM and apparently healthy controls. A secondary objective was to compare the age profile of the two groups and describe the overall thyroid biochemical pattern associated with T2DM in this hospital-based sample.

MATERIALS AND METHODS

Study design and reporting: This hospital-based case-control study compared adults with established T2DM with apparently healthy controls. The manuscript was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology recommendations.[9]

Study setting: Participants with T2DM were recruited from the Medicine outpatient service of Government Medical College and Hospital, Gadwal. Biochemical investigations were performed in the institutional Biochemistry Laboratory. The source protocol also identified academic participation from the Department of Biochemistry, C.A.R. Medical College, Karimnagar.

Participants and group allocation: The analytical sample comprised 50 adults aged 20-70 years. Group I included 25 apparently healthy controls without a known diagnosis of diabetes or thyroid disease. Group II included 25 patients with T2DM attending the outpatient service. Consecutive eligible participants were considered until the prespecified group totals were reached. Formal matching was not documented.

Eligibility criteria: Adults aged 20-70 years with T2DM were eligible for the case group. Controls were adults in the same age range who were considered clinically healthy. Exclusion criteria for both groups were a previous diagnosis of thyroid disease, hepatobiliary disease, pregnancy, and current systemic treatment capable of altering thyroid function, including levothyroxine, antithyroid medicines, glucocorticoids, and oral contraceptives. Participants with incomplete core biochemical measurements were excluded from analysis.

Sample collection and biochemical analysis: After an overnight fast, venous blood was collected using aseptic precautions. Plasma intended for glucose estimation was separated from a fluoride-containing tube, and serum intended for thyroid testing was obtained from a plain collection tube. Samples were centrifuged at approximately 3000 revolutions per minute for 10 minutes and analysed without avoidable delay. Fasting plasma glucose was measured on a fully automated DS-302 analyser (Vector Biotek) using the glucose oxidase-peroxidase enzymatic photometric method. Serum total T3, total T4, and TSH were measured by chemiluminescence immunoassay using.

Study variables and outcomes: The principal exposure was group status, categorized as T2DM or healthy control. Primary outcomes were mean fasting plasma glucose, T3, T4, and TSH concentrations. Age was recorded as a demographic variable. Laboratory values were reviewed for completeness before analysis. Because individual symptom data, diabetes duration, glycated haemoglobin, thyroid autoantibodies, and free thyroid hormone fractions were not available, these variables were not analysed.

Statistical analysis: Continuous variables were summarized as mean ± standard deviation. Between-group differences were evaluated using an independent-samples t test. Two-sided p values below 0.05 were considered statistically significant. Summary comparisons were recalculated from the reported group means, standard deviations, and sample sizes. Statistical processing was performed using.

Ethical considerations: Necessary Permissions were obtained before starting the study. Written informed consent was obtained from every participant before enrolment. Participant confidentiality was maintained during data handling and manuscript preparation.

RESULTS

A total of 50 participants were included in the analysis, with 25 adults in the T2DM group and 25 in the control group. The mean age of patients with T2DM was 39.76 ± 9.39 years, compared with 34.36 ± 12.06 years among controls. The 5.40-year between-group difference was not statistically significant (p=0.084), indicating reasonable comparability for age in this small sample (Table 1).

Table 1. Age distribution of the study groups

Characteristic	Control group (n=25)	T2DM group (n=25)	Mean difference	p-value
Age, years	34.36 ± 12.06	39.76 ± 9.39	5.40	0.084

Values are mean ± standard deviation. T2DM, type 2 diabetes mellitus.

Marked biochemical differences were observed between the groups. Mean fasting plasma glucose was 158.00 ± 50.13 mg/dL in the T2DM group and 94.16 ± 8.87 mg/dL in controls. The mean difference of 63.84 mg/dL was statistically significant (p<0.001). Mean T3 and T4 concentrations were substantially lower among patients with T2DM, whereas mean TSH was considerably higher than in controls (Table 2).

Table 2. Comparison of fasting plasma glucose and thyroid parameters between groups

Parameter	Control group (n=25)	T2DM group (n=25)	Mean difference	p-value
Fasting plasma glucose, mg/dL	94.16 ± 8.87	158.00 ± 50.13	63.84	<0.001
T3, ng/mL	1.22 ± 0.38	0.53 ± 0.47	-0.69	<0.001
T4, nmol/L	95.25 ± 19.38	48.47 ± 21.75	-46.78	<0.001
TSH, mIU/L	1.95 ± 0.78	23.83 ± 12.60	21.88	<0.001

Values are mean ± standard deviation. P values were obtained using independent-samples t tests. T3, triiodothyronine; T4, thyroxine; TSH, thyroid-stimulating hormone; T2DM, type 2 diabetes mellitus.

The mean T3 concentration was 0.53 ± 0.47 ng/mL in the T2DM group versus 1.22 ± 0.38 ng/mL in controls, representing a mean difference of -0.69 ng/mL. Mean T4 was 48.47 ± 21.75 nmol/L in patients with T2DM and 95.25 ± 19.38 nmol/L in controls, a difference of -46.78 nmol/L. Mean TSH was 23.83 ± 12.60 mIU/L in the T2DM group compared with 1.95 ± 0.78 mIU/L in controls, a difference of 21.88 mIU/L. Each thyroid parameter differed significantly between groups (all p<0.001; Table 2). Overall, the direction of change was consistent with a predominantly hypothyroid biochemical profile among patients with T2DM.

DISCUSSION

This case-control study identified a distinct thyroid biochemical pattern among adults with T2DM. Compared with apparently healthy controls, patients with diabetes had significantly lower mean T3 and T4 concentrations and substantially higher mean TSH concentrations. Fasting plasma glucose was also markedly elevated, as expected from the group definition. The age difference was not statistically significant. Taken together, the findings indicate a strong association between T2DM and reduced thyroid function in the studied population, although the cross-sectional comparison does not establish the temporal direction of the relationship. The observed pattern agrees with the broader literature showing that hypothyroid states are the most frequent thyroid abnormalities among people with diabetes. Perros et al. reported a clinically important frequency of thyroid dysfunction in diabetic patients and supported repeated biochemical assessment.[5] In Jordan, Radaideh et al. found thyroid dysfunction in a notable proportion of patients with T2DM, while Papazafiropoulou et al. reported comparable abnormalities among Greek outpatients.[10,11] A more recent case-control study by Khassawneh et al. also demonstrated a higher prevalence of thyroid dysfunction in T2DM than in non-diabetic controls.[12] These studies differ in age distribution, iodine exposure, assay thresholds, and case definitions, but their collective direction is consistent with the present results.

Several mechanisms could explain lower circulating T3 and T4 with increased TSH in T2DM. Insulin resistance and hyperinsulinaemia influence thyroid tissue growth and hormone metabolism, while sustained hyperglycaemia can alter hypothalamic-pituitary signalling and reduce peripheral deiodination of T4 to T3.[2,3] Systemic inflammation, calorie imbalance, renal impairment, medication exposure, and non-thyroidal illness can further modify thyroid tests. The meta-analysis by Han et al. demonstrated a significant association between subclinical hypothyroidism and T2DM, and the systematic review by Hadgu et al. confirmed that thyroid dysfunction remains common across diverse diabetic populations.[6,7] Thus, the abnormalities recorded here are biologically plausible and consistent with pooled evidence. Not all studies show an increased prevalence of categorical thyroid disease. In a prospective observational investigation, Iwakura et al. found altered thyroid hormone relationships in T2DM but no clear excess of diagnosed thyroid dysfunction or thyroid autoantibody positivity compared with controls.[13] This difference emphasizes the importance of distinguishing primary thyroid disease from metabolic changes in hormone conversion. Likewise, prospective population data indicate that lower thyroid function can precede incident prediabetes or T2DM, suggesting that the relationship operates in both directions.[14] Clinically, the results support thyroid testing when a patient with T2DM has poor glycaemic control, unexplained dyslipidaemia, weight change, fatigue, treatment-resistant symptoms, or other features suggestive of thyroid disease. Routine interpretation should include repeat testing, free hormone measurements, medication review, renal and

hepatic status, and thyroid autoantibodies when indicated. The pronounced group differences in this study warrant confirmation using larger, multicentre samples with individual-level data and standardized diagnostic categories.

LIMITATIONS

This study had a small, single-centre sample and a case-control design, which restrict external validity and causal interpretation. Individual data on diabetes duration, glycated haemoglobin, body mass index, treatment, renal status, thyroid autoantibodies, and free thyroid hormone fractions were unavailable. Residual confounding therefore remains substantial. The absence of longitudinal follow-up also prevents assessment of whether thyroid abnormalities preceded diabetes, followed poor glycaemic control, or changed after treatment.

CONCLUSION

Adults with type 2 diabetes in this hospital-based study had substantially higher fasting plasma glucose and TSH concentrations, together with markedly lower T3 and T4 concentrations, than healthy controls. This biochemical pattern indicates a strong association between type 2 diabetes and reduced thyroid function, particularly a hypothyroid profile. Because unrecognized thyroid dysfunction can complicate metabolic control and cardiovascular risk assessment, thyroid evaluation should be considered in diabetic patients with poor glycaemic control, suggestive symptoms, longer disease duration, or unexplained lipid abnormalities. Larger multicentre studies incorporating free thyroid hormones, thyroid autoantibodies, glycated haemoglobin, renal function, and longitudinal follow-up are required to define clinically effective screening strategies and outcomes in routine practice.

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