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Original Article

The mediators and predictors of higher values of thyroid-stimulating hormone in type 2 diabetes mellitus: a cross-sectional study

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Abstract

Subclinical hypothyroidism associated with a higher level of thyroid-stimulating hormone (TSH) is commonly reported in type 2 diabetes mellitus (T2DM). Multiple causes, rather than a specific cause, have been reported for this association. This study aims to investigate some biomarkers as mediators or predictors of the higher levels of TSH in T2DM. This cross-sectional study included 150 diabetic patients. The patients were grouped according to their TSH levels into Group I (0–4 µIU), Group II (4.1–8.0 µIU), and Group III (>8.0 µIU). Anthropometric measurements, glycemic and lipid indices, thyroid hormones, and inflammatory biomarkers (including C-reactive protein and interleukin 6) were determined. The mediation (indirect effect) was assessed using the Sobel test, and prediction was evaluated using logistic regression and Receiver Operating Characteristic tests. Groups II and III showed significantly higher values of these biomarkers. None of these biomarkers has an indirect (mediation) effect on the high levels of TSH. Duration of the disease, triglyceride-glucose index, interleukin-6, and stress hyperglycemia ratio are significant predictors. These predictors have areas under the curves of 0.810, 0.805, 0.869, and 0.562, respectively. We conclude that specific biomarkers can predict higher TSH levels, but they do not act as mediators.

Keywords: type 2 diabetes mellitus, thyroid-stimulating hormone, mediation effect, predictors, triglyceride-glucose index, interleukin-6

Introduction

Type 2 diabetes mellitus (T2DM) is a common chronic endocrine disease commonly associated with thyroid gland dysfunction [1]. Both hyperthyroidism and hypothyroidism are concomitant illnesses with T2DM, and thyroid dysfunction in terms of hypothyroidism could be a risk for developing prediabetes [2]. Subclinical or clinical hyperthyroidism was reported in 1.13% and 3.16%, respectively, among 1677 patients, while higher percentages of 4.89% and 9.3% were reported with subclinical and overt hypothyroidism [3]. A systematic review with meta-analysis, which included 12 prospective studies, found that 17% of participants with high thyroid-stimulating hormone (TSH) are at risk for developing T2DM. Such an association is not linear, but it appears to be a J-shape [4]. The level of TSH in T2DM is studied thoroughly as a diagnostic and/or prognostic factor in many associated pathological conditions. The upper normal level of TSH (up to 5 µIU) was not significantly related to incident T2DM [3]. However, a higher TSH (>4.2 µIU) in diabetic patients was strongly associated with microvascular complications, such as diabetic kidney disease [5]. A higher level of TSH is associated with a higher level of glycosylated hemoglobin (HbA1c%), which may indicate that the TSH level is highly elevated in uncontrolled diabetes [6]. One study summarized the risk factors associated with thyroid

dysfunction in T2DM, which include female gender, central obesity, duration of diabetes, microvascular neuropathy, and an HbA1c level of >7% [7]. An association between TSH levels and serum cholesterol levels was reported in euthyroid T2DM patients [8]. There is evidence that TSH has a role in diseases characterized by low-grade inflammation, such as T2DM, where a significantly higher TSH level is associated with elevated inflammatory markers, including interleukin (IL)-6, while a lower TSH level is associated with a higher IL-8 in euthyroid T2DM [9]. In autoimmune thyroiditis, a higher mean TSH level (7.6 IU/L) is associated with a higher mean IL-6 level (9.4 pg/mL) [10]. A recent study reveals a significant positive correlation between the triglyceride-glucose (TyG) index and TSH in the general population, suggesting that elevated TSH levels are associated with impaired glucose homeostasis [11]. Thakur *et al*. observed that the TyG index is significantly increased in subclinical hypothyroidism, but it did not correlate with thyroid hormones [12]. Another confirmatory study found a significant correlation between TSH and the TyG index in women, indicating a gender-based effect [13]. Others reported that men and women who have a higher TyG index are at risk of subclinical hypothyroidism, and this risk disappears in women at menopause [14]. The complex relationship between TSH and various biomarkers in T2DM leads us to fill the gap that these biomarkers are mediators or predictive of the risk of high TSH levels in T2DM by analyzing the glycemic indices, inflammatory indices, and the TyG index. Therefore, this cross-sectional cohort study aimed to identify the biomarkers that mediate or predict thyroid dysfunction in T2DM patients.

Material and methods

Setting and design

This cross-sectional study was conducted at the College of Medicine, the University of Diyala, in collaboration with Ishtar Medical Institute between January and June 2025. One hundred fifty diabetics were randomly recruited using random tables.

Ethical approval

The Institutional Ethics Committee approved this study (No. 2025MSN 951), according to the principles of the Declaration of Helsinki. There is no interference with the therapeutic regimen (either adding or omitting medicine), and the patients were free to withdraw from the study.

Participants

Patients with T2DM of both sexes, aged 35 years or older, who were treated with oral antidiabetics were eligible. Patients with a previous history or preexisting thyroid disorders or using thyroid-related hormones; insulin therapy users; those with type 1 diabetes or latent onset diabetes (positive glutamic acid decarboxylase antibody test), acute infections, or suspected surgical conditions within four months; those with autoimmune diseases, such as systemic lupus erythematosus, or skeleto-muscular pathological conditions; those using non-steroidal anti-inflammatory or steroidal therapy; those with terminal illness; and pregnant or breastfeeding women were excluded. Each patient was asked about his/her duration of diabetes and current medications, and clinically examined. The patients were grouped according to their TSH levels into:

- Group I: patients with TSH levels up to 4 μIU (a normal limit).
- Group II: patients with TSH levels ranging between 4.1 and 8.0 μIU (a moderate increase level);
- Group III: Patients with TSH levels more than 8 μIU (a higher level).

Primary and secondary outcomes

The primary outcome is TSH, and the predictor variables were duration of the disease, glycemic indices, inflammatory biomarkers (including CRP and IL-6), and triglyceride-glucose index. The secondary outcomes include age, sex, triiodothyronine (T3), thyroxine (T4), anthropometric, and lipid indices.

Measurements

I. Anthropometric measurements

Anthropometric measurements, including body weight (kg), height (cm), and waist circumference, were taken. The body mass index (kg/m^2) is calculated by dividing the weight as the numerator and the square of the height as the denominator. The conicity index was calculated using the following equation:

$$\text{Conicity index} = \frac{Waist\ circumference}{0.109\sqrt{\frac{Weight}{Height}}}$$

II. Blood sampling, biochemical, and immunological measurements

Blood samples were obtained as routine laboratory investigations for the clinical assessment of the patients. A fasting morning blood samples were taken, and separated into two portions; the first was centrifuged at 3,000 rpm for 15 minutes to separate the sera for the determination of serum glucose, lipid profile, thyroid hormones, CRP and IL-6. The second portion was kept in the EDTA tubes for the determination of the HbA1c (%). The serum glucose, total cholesterol, triglyceride, high-density lipoprotein-cholesterol, thyroid-related hormones (TSH, T3, and T4) and CRP were determined in the clinical laboratories at the diabetic centers. Non-high-density lipoprotein-cholesterol (Non-HDL-C), TyG index, and stress hyperglycemic ratio (SHR) were determined by using the following equations:

$$\text{Non-HDL-C} = \text{Total cholesterol } \textit{minus} \text{ ([Triglyceride} \times 0.2] + \text{HDL-C)}$$

$$\text{TyG index} = \text{Ln}\frac{Triglyceride\left(\frac{mg}{dl}\right) \times glucose\left(\frac{mg}{dl}\right)}{2}$$

$$\text{SHR} = \frac{Current\ blood\ glucose}{estimated\ blood\ glucose}$$

where the estimated blood glucose is equal to 28.7 multiply by HbA1c(%) minus 46.7 [15].

The concentration of interleukin-6 was measured by using the enzyme-linked immunosorbent assay (ELISA) technique according to the instructions of the manufacturer by using a human IL-6 ELISA kit (Elabscience, Texas, USA).

Statistical analyses

Sample size estimation was performed using the following equation based on 19,250 diabetic patients in the study area and a 2% expected high TSH [16].

$$\text{Sample size } (n) = \frac{N \times Z^2 \times p(1-p)}{E^2 \times (N-1) + Z^2 \times p(1-P)} = 150.3$$

where: N is the population size = 385 for 95% confidence, Z score = 1.96; the estimated population proportion, p = 20% = 0.2 (β error); the margin of error, E = 0.05 (α error).

The results are presented as numbers, percentages, medians, interquartile ranges (IQRs), standard errors, and 95% confidence interval. The differences between medians were analyzed using the independent samples Kruskal-Wallis test. The collinearity diagnostic test was used to calculate the tolerance and variance inflation factor (VIF) to show the impact of a sample size on the results. Mediation analysis was done using the Sobel test to show the effect of independent variables (mediators) on the level of TSH (as a dependent variable). A logistic regression analysis was performed to identify predictors of higher levels of TSH. Receiver operating characteristic (ROC) curves were used to assess the predictive value of increased duration of disease, TyG index, IL-6, and SHR, with the area under the curve (AUC) calculated (including the sensitivity, specificity, positive predictive value, negative predictive value, and Youden's index) at a cutoff value of each predictor. SPSS-IBM compatible software version 26.0 (Chicago, Illinois, USA) was used to perform the statistical analyses, with a p-value of ≤0.05 considered statistically significant.

Results

Table 1 shows the characteristics of the participants. The sex distribution, age, and family history of diabetes did not significantly differ among the groups. The frequency of smokers in Group III is significantly higher than that in Group II. The levels of TSH were significantly higher as the duration of the disease prolonged. The results of the glycemic indices showed significant differences between groups, irrespective of the TSH levels. The thyroid hormones (T3 and T4) progressively and significantly decreased as the TSH levels increased. Table 2 shows that participants with a TSH level higher than 8 μIU/mL have significantly higher mean values of the anthropometric indices, atherogenic lipids, TyG index, and IL-6 than those in Groups I and II. The mean value of HDL-C is significantly less than that in Groups I and II, while the mean value of CRP showed a non-significant difference between groups. Table 3 shows that the associated variables for thyroid dysfunction and type 2 diabetes do not significantly mediate the higher levels of TSH in diabetic patients, as the p-values (Sobel test statistics) are >0.05 for duration of the disease, conicity index, stress hyperglycemia ratio, CRP, and IL-6.

Table 4 shows the results of the logistic regression and collinearity statistics. The duration of the disease,

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Table 1: Characteristics of the participants.

Variables	Group I (n=29)	Group II (n=45)	Group III (n=76)	P1	P2	P3
Sex (Female: Male)	17:12	32:13	59:17	0.267	0.051	0.644
Age (year)	50 (49, 55.5)	50 (48, 55)	51 (49, 55)	0.308	0.308	0.308
Smoking						
None	14	26	29	0.641	0.889	0.036
Current	15	19	47			
Family history of diabetes	8	13	20	0.903	0.895	0.094
Duration of diabetes	2 (1, 3)	3 (2, 5)	4 (4, 5)	0.005	<0.001	0.001
Glycemic indices						
Random blood glucose (mg/dL)	207 (187, 232.5)	188 (167, 199.5)	208 (188, 238)	0.002	0.874	<0.001
Glycosylated hemoglobin (%)	8.1 (7.4, 8.35)	8.5 (8, 8.75)	8.5 (8.2, 8.8)	0.001	<0.001	0.864
Stress hyperglycemia ratio	1.157 (1.044, 1.249)	0.943 (0.874, 1.039)	1.083 (0.931, 1.242)	<0.001	0.032	<0.001
Thyroid hormones						
TSH (µIU/mL)	0.29 (0.21, 0.37)	7.2 (6.5, 7.5)	9.4 (8.5, 10.3)	<0.001	<0.001	<0.001
Thyroxine (µg/dL)	130 (108.6, 140)	75.6 (69.1, 111)	62.4 (54.9, 70.1)	<0.001	<0.001	<0.001
Triiodothyronine (ng/dL)	2.9 (2.5, 3.3)	1.1 (1.04,2.65)	0.95 (0.89, 1.05)	<0.001	<0.001	<0.001

Note: The results are presented as median (IQR). P-values were calculated using an independent samples Kruskal-Wallis test (for comparison between groups; P1 – between Group I and II, P2 – between Group I and III, and P3 – between Group II and III), and a chi-square test for categorized data.

Table 2: Anthropometric, lipid profile measurements, ratios and indices, and inflammatory biomarkers.

Variables	Group I (n=29)	Group II (n=45)	Group III (n=76)	P1	P2	P3
Body mass index (kg/m^2)	34.7 (33.5, 36.4)	34.7 (33.5, 36.4)	37.7 (36.5, 38.8)	0.692	<0.001	<0.001
Waist circumference (cm)	86 (81, 87.5)	86 (81, 87.5)	98 (94, 100)	0.014	<0.001	<0.001
Conicity index	1.05 (1.01,1.07)	1.05 (1.01, 1.07)	1.13 (1.1, 1.15)	0.011	<0.001	<0.001
Triglycerides (mg/dL)	136 (125, 142.5)	274 (137.5, 318)	290 (269, 316)	<0.001	<0.001	0.032
Total cholesterol (mg/dL)	157 (146, 174.5)	241 (177.5, 274)	289 (257, 311)	<0.001	<0.001	<0.001
HDL-C (mg/dL)	60 (58, 61.5)	49 (39.5, 58)	39 (36, 42)	<0.001	<0.001	<0.001
Non-HDL-C (mg/dL)	68.6 (59.3, 84.3)	135 (89.6, 171.4)	194.8 (162.6, 213.8)	<0.001	<0.001	<0.001
Triglyceride-glucose index	9.6 (9.43, 9.68)	10.07 (9.47, 10.22)	10.34 (10.13, 10.49)	0.003	<0.001	<0.001
Interleukin-6 (pg/mL)	376.9 (330.1, 389.1)	332.9 (312.9, 372.0)	453.3 (389, 538.3)	0.078	<0.001	<0.001
CRP (mg/L)	4.7 (4.55, 5.4)	4.3 (3.8, 4.6)	4.9 (4.3, 5.4)	<0.001	0.631	<0.001

Note: The results are presented as median (IQR). P-values were calculated using an independent samples Kruskal-Wallis test (for comparison between groups; P1 – between Group I and II, P2 – between Group I and III, and P3 – between Group II and III). HDL-C – high-density lipoprotein-cholesterol, CRP – C-reactive protein.

Table 3: The results of the Sobel test show the indirect effect of the mediators on the changes of the thyroid-stimulating hormone in type 2 diabetes.

Mediators	Indirect effect		Z score	p-value (Sobel test statistic)
	Indirect effect±SE	95% confidence interval		
Duration of disease	0.0097±0.021	-0.0305, 0.0499	0.473	0.636
Conicity index	1.18±1.26	-1.286, 3.645	0.938	0.348
Stress hyperglycemia ratio	1.368±1.37	-1.318, 4.053	0.998	0.318
Triglyceride-glucose index	0.311±0.316	-0.309, 0.931	0.984	0.325
C-reactive protein	0.168±0.172	-0.168, 0.504	0.981	0.326
Interleukin-6	0.0013±0.0014	-0.0013, 0.0039	0.984	0.325

TyG index, IL-6, and SHR are significant predictors for high TSH levels in diabetic patients. These findings suggest that anthropometric measurements and CRP (as a systemic, nonspecific inflammatory marker) do not significantly predict higher levels of TSH in T2DM.

Figure 1 shows the AUC of the significant predictors mentioned in Table 3. The AUC of the duration of diabetes is 0.810, p<0.001 at a cutoff value of four years (sensitivity: 93.2%, specificity: 37.1%, PPV: 67.8%, NPV: 79.3%, and Youden's index: 0.303). A comparable significant AUC of the TyG index (AUC: 0.805, p=0.001) was observed at a cutoff value of 9.4. The sensitivity is 85.4%, specificity: 33.3%, PPV: 93.3%, NPV: 17.2%, and Youden's index: 0.187. The AUC of the IL-6 is the highest one, which accounted for 0.869 (p<0.001) at a cutoff value of 400 pg/mL. The sensitivity, specificity, PPV, NPV, and Youden's index are 90.8%, 24.1%, 48.8%, 79.3%, and 0.115, respectively. The AUC of SHR is non-significantly (p=0.202) lower than the other predictors, which accounted for 0.562 at a cutoff value of 1.00. The sensitivity, specificity, PPV, NPV, and Youden's index are 74.4%, 10%, 20.7%, and 79.3%, respectively. These results indicate that the predictors are sensitive but not specific for predicting the higher levels of TSH.

Discussion

The results of this study showed that the duration of diabetes, conicity index, and TyG index are significant predictors, but they do not act as mediators for the development of subclinical high TSH level. A recent study demonstrates that elevated TSH as a marker of subclinical hypothyroidism in individuals with impaired glucose tolerance is a transient condition because the significant decrease in HbA1c% is associated with a decline in TSH level and a reversion to the normal level [17]. Therefore, the higher HbA1c% value

Table 4: The results of logistic regression using four models to identify the predictors of higher values of thyroid-stimulating hormone in diabetic patients.

Model	Predictors	R	F-value	P-value	β-coefficient (C.I.)	p-value	Collinearity statistic	
							Tolerance	VIF
Baseline	Duration	0.422	32.0	<0.001	1.160 (0.755, 1.565)	<0.001	1	1
Model 1	TyGI	0.607	42.8	<0.001	5.066 (3.560, 6.572)	<0.001	0.642	1.558
Model 2	hs-CRP	0.610	28.0	<0.001	-0.345 (-1.061, 0.370)	**0.342**	0.975	1.026
Model 3	IL-6	0.644	25.6	<0.001	0.011 (0.004, 0.017)	0.001	0.598	1.673
Model 4	SHR	0.675	24.2	<0.001	-4.501 (-7.169, -1.833)	0.001	0.805	1.243
Model 5	Conicity	0.675	20	<0.001	0.879 (-11.578, 13.337)	**0.899**	0.445	2.247

Note: A VIF >10 or >5 in some cases is a statistical problem; a tolerance <0.1 or in some cases <0.2 is a problem. VIF >10 is a sign of high multicollinearity. P-values were calculated using analysis of variance test, and a collinearity diagnostic test (a value of VIF >10 indicates an impact of the sample size on the results). R – correlation factor, and a F-value – Fixation value.

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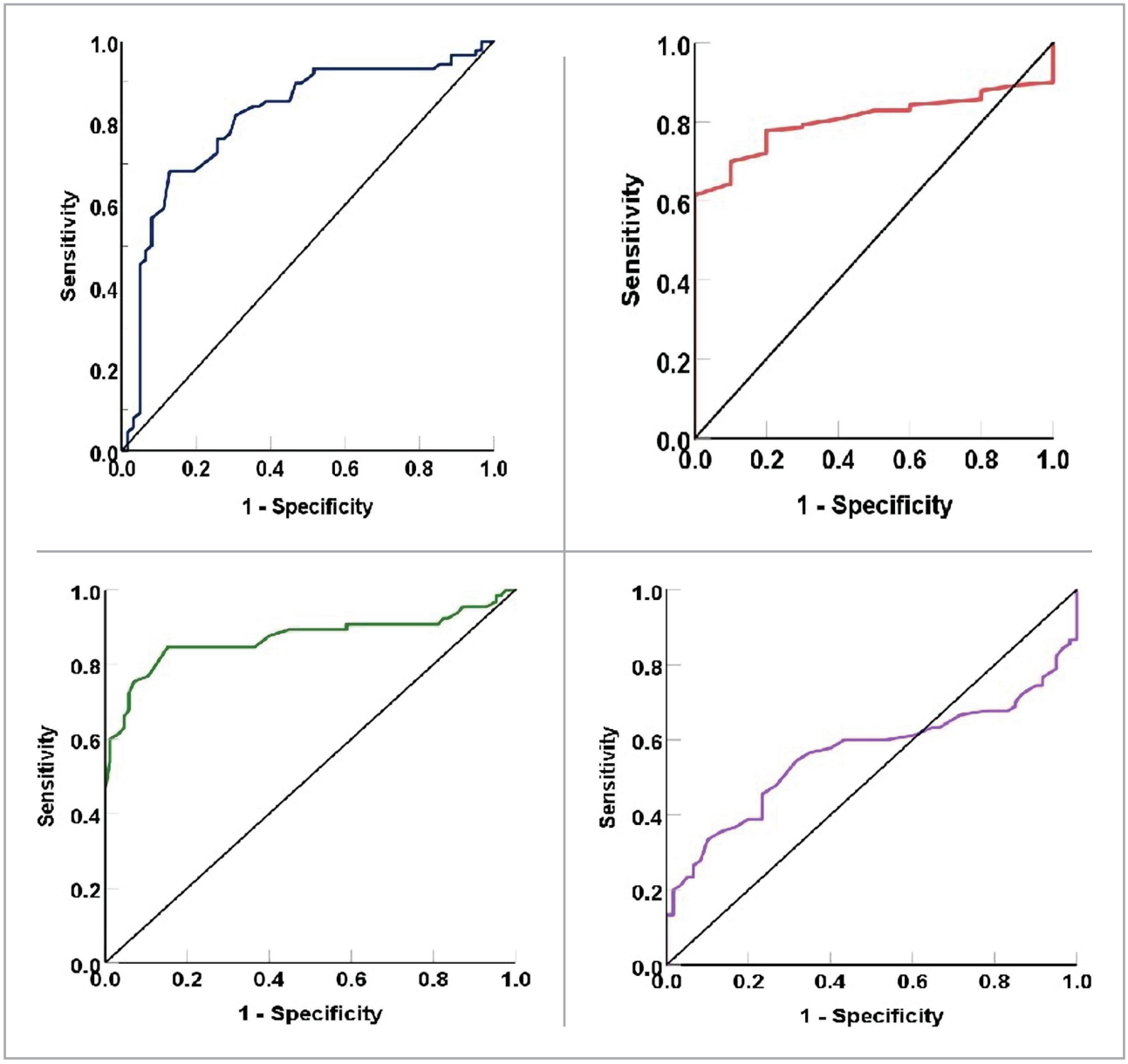

Figure 1: The area under the curve of the thyroid-stimulating hormone using the predictors: duration of the disease (upper left corner), triglyceride-glucose index (upper right corner), interleukin-6 (lower left corner) and stress hyperglycemia ratio (Lower right corner).

is associated with an above-normal limit of TSH in 121 out of 150 (80.7%). Other studies demonstrated the predictors of subclinical hypothyroidism in complicated T2DM. In diabetic retinopathy, subclinical hypothyroidism is associated with a longer duration of the disease and poor glycemic control, as our study demonstrates this finding [18]. Another study found that poor glycemic control (higher HbA1c% and HOMA-IR values) in diabetic neuropathy is a significant predictor for higher TSH levels [19]. The TyG index is a predictor for many pathological conditions related to diabetes, *e.g.*, cardiovascular events [20, 21]. Our study agreed with others who reported a significant positive association between the TyG index and TSH in the general population [22]. The TyG index impacts the indirect (mediation) effect on the association between thyroid hormones (T3 and T4) and metabolic dysfunction-associated fatty liver disease [23]. In the current study, the TyG index does not impact mediation effects on the association between TSH (a pituitary hormone) and T2D. The second significant predictor of higher TSH values is IL-6. A recent study demonstrates that the predictive values of IL-6 and TSH are similar in predicting carotid plaques in patients with carotid artery disease [24].

There is evidence that an alteration in the IL-6 level in hypothyroidism resulted from radioactive iodine (I-131) [25]. Therefore, the results of the current study support previous studies indicating an association between

IL-6 and TSH and that IL-6 is a significant predictor of higher TSH levels. An experimental study using cadmium as a metal to disrupt thyroid hormones showed increasing IL-6 levels, indicating that a hypothyroid state has an indirect (mediation) effect on the higher IL-6 levels [26]. Higher levels of IL-6 do not mediate the higher levels of TSH in T2D patients in our study, which confirms a previous study that higher TSH indirectly impacts increases in the inflammatory biomarkers and not the opposite. The SHR is an inferior biomarker compared with the duration of the disease, the TyG index, and IL-6 to predict a significantly higher TSH level. The relationship between hyperglycemia and thyroid gland dysfunction is complex because many modifiable and non-modifiable confounding factors are involved, and this explains why SHR is a weak predictor [27].

The fourth predictor is the duration of the disease, which is involved in the prediction of many pathological conditions in T2D, but it does not mediate the cause of high TSH levels. The strength of the study is that the direct and indirect impact of glycemic indices, inflammatory biomarkers, TyG index, and duration of the disease are studied at the same time and prove that these factors are associated predictors rather than mediators. The limitations of the study are that the data were collected from a single center, and the sample size of patients with normal TSH levels is small compared with those having higher levels. Even though these limitations will not bias the results.

Conclusion

Subclinical hypothyroidism and type 2 diabetes are commonly associated. Therefore, many markers that relate to the diabetes or inflammatory or immunological process significantly predict high levels of thyroid-stimulating hormone. Among these markers, the duration of type 2 diabetes mellitus, interleukin-6, and triglyceride-glucose index are significant predictors but not mediators for the significantly associated higher thyroid-stimulating hormone. Therefore, the detection of subclinical hypothyroidism in diabetic patients is not mediated by inflammatory or lipid profiles or the longstanding duration of diabetes.

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Conflict of interest

The authors declare no conflict of interest.

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