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ORIGINAL STUDY

Thyroid Diseases as a Risk of Type 2 Diabetes Mellitus

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Abstract

Objectives: **To evaluate thyroid disease as a risk of type two diabetes.**

Background: **Thyroid dysfunction and diabetes mellitus are closely linked. Thyroid diseases affect the secretion, action, and metabolism of insulin so induce insulin resistance and lead to diabetes.**

Methods: **This cohort study involved 120 Egyptian patients divided into 49 patients with hypothyroidism, 37 patients with hyperthyroidism, and 34 subjects in the control group and we noticed their effects on insulin metabolism. Glycated hemoglobin A1c; Homeostatatic Model of Assessment of insulin resistance (HOMA-IR), thyroid antibodies were measured from plasma samples using an enzyme −linked immunosorbent assay (ELISA) and other laboratory methods.**

Results: **Type 2 diabetes risk was increased in thyroid disease patients, especially in the duration of half year to one year after complaining of thyroid dysfunctions. Mean of HbA1c was increased significantly in thyroid dysfunction groups than in the control group, correlation of hyperthyroidism to HbA1c was 6.20 (P value = 0.004) and correlation of hypothyroidism to HbA1c was 6.50 (P value = 0.004). HBA1c level was increased in hyperthyroid patients by 0.76 and in hypothyroid patients by 1.05 compared to normal (P value < 0.05).**

Conclusion: **For better treatment and early diagnosis of diabetes, blood glucose tests should be done in patients with thyroid dysfunction after the prime diagnosis of thyroid disease.**

Keywords: Autoimmune thyroid, Hyperthyroidism, Hypothyroidism, thyroid disease, Type 2 diabetes

1. Introduction

Thyroid disease is widespread in community, and prevalence of thyroid dysfunction increases with age. Modern assays of assessment of thyroid dysfunction give the advantages of being cheap and reliable. Certain high-risk groups, like neonates and old persons are indicated for screening for thyroid dysfunction [1].

Diabetes mellitus (DM) is also common in the community. The widespread prevalence of this disease in adults had increased from 4.7% in 1980 to 8.5% in 2020. National Health and Nutrition Examination Survey (NHANES) III reports that about 14% of the US adult population complain from either DM or impaired fasting blood glucose level [2].

Although hyperthyroidism is accompanied with improved insulin secretion in persons with prediabetes. However, it also has a deleterious effect on the metabolism of glucose in patients with DM [3].

Thyroid hormone deficiency leads to impairment of peripheral insulin sensitivity and glucose metabolism and treatment of hypothyroidism leads to improvement of insulin sensitivity [4].

Good control of blood glucose and thyroid hormone levels may help in prevention of DM in patients with thyroid disease [5].

The aim of our study was to evaluate if thyroid disease is a risk of type 2 DM (T2DM).

2. Subjects and methods

This cohort study was conducted on 120 participants who were selected from inpatient and the outpatient clinic of the department of internal medicine at Menoufia University Hospitals from June 2021 to March 2022. All participants included in

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this study gave informed consents, the study was approved by the Ethics and Research committee of the Faculty of Medicine, Menoufia University. The subjects of the study were subdivided into the following groups: **Group 1: 37 patients** with newly diagnosed hyperthyroidism, **group 2:** 49 patients with newly diagnosed hypothyroidism, **group 3 (control group):** 34 of volunteer healthy subjects. Patients were excluded if they had been diagnosed with hyperthyroidism, hypothyroidism or if they are less than 18 years old. Patients with history of diabetes, genetic defects, pancreatitis, or pancreatectomy were also excluded. All patients were subjected to: Full history taking specially: Age, Gender, history of thyroid dysfunction and its duration, Clinical examination of the neck for any growths or enlargement of the thyroid, Thyroid stimulating hormone (TSH), Free tri-iodo-thyronine (T3), free thyroxine (T4) and Thyroid antibodies: TSH receptor antibody (TRAB), Anti Thyroglobulin antibodies (anti-Tg Ab), Fasting blood glucose (FBG) and 2-h post-prandial blood glucose (2hrPPBG) using the oral glucose tolerance test, HbA1c and Homeostatatic Model of Assessment for insulin resistance (HOMA-IR).

TRAB ELISA kits (Medical supply company! Mulhuddart; Dulbin) was used to measure serum TRAB.

Human Anti-Tg igG ELISA Kit (Epitope Diagnostics company, San Diego, U.S.A.) was used to measure serum anti-Tg Ab.

TSH Enzyme immunoassay test kit (PerkinElmer Health Sciences Inc, Hayward, U.S.A) was used to measure serum TSH.

Free T3 ELISA Kit (AutoBio Diagnostics Company, Brussels, Belgium) was used to measure serum Free T3.

Free T4 ELISA Kit (AutoBio Diagnostics Company, Brussels, Belgium) was used to measure serum Free T4.

HbA1c Reagent kit (Axis-Shield Diagnostics Ltd; Dundee; United Kingdom) was used to measure HbA1c.

Glucose oxidase method was used to measure FBG, as described by Trinder, and 2hrPPBG was measured by using oral glucose tolerance test, using commercial kit Biolabo Reagents, France.

HOMA-IR equation was used to measure insulin resistance from fasting serum glucose and insulin using the Oxford HOMA Calculator.

Clinical, Laboratory data of the cases was tabulated.

2.1. Statistical analysis

Our collected data was coded and tabulated, then it was introduced to a PC using Statistical package for Social Science (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp). Suitable analysis of data was done according to the type of data obtained for each parameter. Quantitative data was subjected to descriptive analysis using median and interquartile range for abnormally distributed variables and mean and standard deviation for normally distributed variables. For qualitative categorical variables, frequency and percentage were applied. Analytical statistics One-way ANOVA/Kruskal−Wallis tests were performed to estimate the change in numerical variables. Tukey HSD/Dunn tests were performed to assess the difference among groups. Chi-squared test was performed to estimate the change in categorical variables.

Spearman correlation was performed to estimate the linear association between diabetic and thyroid tests. Linear regression was performed to predict factors associated with mean levels of HBA1cLogistic regression was performed to predict factors associated with diabetes. A two-tailed test using a level of significance for analysis at $p \leq 0.05$ was used for statistical analysis.

3. Results

As regard the body mass index (BMI), the median of BMI of the hyperthyroid group was 24.6 kg/m^2 and that of the hypothyroid group was 35.15 kg/m^2 and that of control group was 26.6 kg/m^2 with significant difference between the three groups (P value < 0.001) (Table 1).

As regard goiter, it was presented in 70.27% of patients in hyperthyroid group, in 34.69% of patients in hypothyroid group and in 2.94% of patients in control group, with significant difference between the three studied groups (P value < 0.001) (Table 2).

The median of TSH of hyperthyroid group was 0.03 mIu\Land that of hypothyroid group was 9.4 mIu\Land that of control group was 1.74 mIu\Lwith significant difference between the three studied groups (P value < 0.001). The median of anti-Tg Ab of hyperthyroid group was 182.9 IU\mL and that of hypothyroid group was 85.5 IU\mL and that of control group was 62.6 IU\mL with significant difference between the three studied groups (P value < 0.001). The median of TRAB of hyperthyroid group was 3.23 mIu\mL and that of hypothyroid group was 1.46 mIu\mL and that of control group was 1.2 mIu\mL with significant difference between the three studied groups (P value < 0.001), the median of HbA1c of hyperthyroid group was 6.2% and that of hypothyroid group was 6.5% and in control group was 5.3% with significant difference

Table 1. Baseline Characteristics of the studied groups.

Dependent: disease	Unit	Normal ($n = 34$)	Hyperthyroidism ($n = 37$)	Hypothyroidism ($n = 49$)	P value
Age (Years)	Mean (SD)	38.88 (11.77)	41.05 (11.95)	42.45 (10.90)	$P = 0.382$
Sex	Female	20 (58.82)	33 (89.19)	43 (87.76)	$P = 0.001$
					$P1 = 0.011$
					$P2 = 0.011$
					$P3 = 0.999$
	Male	14 (41.18)	4 (10.81)	6 (12.24)	
Height (Cm)	Median (IQR)	165.00 (160.00–169.75)	160.00 (160.00–165.00)	160.00 (159.00–162.00) †	$P = 0.035$
					$P1 = 0.325$
					$P2 = 0.033$
					$P3 = 0.250$
Weight (kg)	Mean (SD)	76.29 (14.76)	67.54 (10.76) †	88.16 (14.87) †*	$P < 0.001$
					$P1 = 0.02$
					$P2 = 0.005$
					$P3 < 0.001$
BMI (kg/m^2)	Median (IQR)	26.60 (25.39–29.29)	24.60 (23.43–26.42)	35.15 (31.22–37.73) †*	$P < 0.001$
					$P1 = 0.06$
					$P2 < 0.001$
					$P3 < 0.001$

BMI, basal metabolic index; Cm, centimeter; IQR, interquartile range; Kg, kilogram; kg/m^2, kilogram/meter^2; n, number; *P*.value, probability value; SD, standard deviation; P: comparison between the 3 group.
*P*1: comparison between hyperthyroid and normal group.
*P*2: comparison between hypothyroid and normal group.
*P*3: comparison between hyperthyroid and hypothyroid group.
P.value < 0.05 is significant, *P*.value < 0.001 is highly significant.
Age and weight are shown as mean (SD), analyzed by ANOVA followed by the Tukey HSD test.
Height and BMI are shown as median (IQR), analyzed by the Kruskal Wallis test followed by the Dunn test.
Other data are shown as count (percent), analyzed by Chi-squared test compared to the normal group, * compared to the hyperthyroid group†.
Post hoc analysis of baseline characteristics by Dunn test/Tukey HSD test/Fisher test.

between the three studied groups (*P* value = 0.004) (Table 2).

Median of FBG of hyperthyroid group was 130.00 mg\dL and that of hypothyroid group was 127.00 mg\dL and that of control group was 92.3 mg\dL with significant difference between the three studied groups (*P* value < 0.001). Median of HOMA-IR of hyperthyroid group was 1.7 and that of hypothyroid group was 1.6 and that of control group was 1.2 with significant difference between the 3 groups (*P* value < 0.001) (Table 2).

As regard to thyroid and diabetic Laboratory tests, there was a positive correlation between free T3 and FBG (CI = 0.017), free T3 and 2 hrPPBS (CI = 0.03), TSH and HbA1c (CI = 0.05), TSH and HOMA-IR (CI = 0.12), anti-Tg Ab and HbA1c (CI = 0.2), anti-Tg Ab and FBG (CI = 0.13, anti-Tg Ab and 2hrPPBS (CI = 0.08), anti-Tg Ab and HOMA-IR (CI = 0.2), TRAB and HbA1c (CI = 0.06), TRAB and FBS (CI = 0.09), TRAB and 2hrPPBG (CI = 0.07), TRAB and HOMA-IR (CI = 0.2), while there was a negative correlation between free T3 and HbA1c (CI = −0.05), free T3 and HOMA-IR (CI = −0.07), free T4 and HbA1c (CI = −0.12), free T4 and FBS (CI = −0.08), free T4 and 2hrPPBS (CI = −0.07), free T4 and HOMA-IR (CI = −0.14), TSH and FBG (CI = −0.03), TSH and HOMA-IR (CI = −0.02) (Table 3).

In multivariate analysis, we found an increased HbA1c level by 0.002%for each increase in anti-Tg Ab (*P* < 0.05), 0.013%for each increase in FBS (*P* < 0.05), 0.013% for each increase in 2hrPPBS (*P* < 0.05). A decreased HbA1c level by 0.021%for each increase in TRAb (Table 4).

In multivariate logistic regression analysis, we found decreased odds of being a diabetic with increased free T4 (OR: 0.86, *P* < 0.05), increased odds of being a diabetic with hyperthyroid patients compared to normal (OR: 55.63, *P* < 0.05) (Table 5).

Hyperthyroid patients have 3.36 times the risk of being diabetic compared to normal. Hypothyroid patients have 3.12 times the risk of being diabetic compared to normal (Table 6).

4. Discussion

In this study we found that the mean age of the studied group with hyperthyroidism was 41.05 +_11.95 years old and with hypothyroidism was 42.45 ± 10.90 years old, in contrast with Veltri F et al.

Table 2. Clinical Characteristics of the studied groups.

Dependent: disease	Unit	Normal ($n = 34$)	Hyperthyroidism ($n = 37$)	Hypothyroidism ($n = 49$)	*P* value
Goiter	No	32 (94.12)	11 (29.73)	32 (65.31)	$P < 0.001$ $P1 < 0.001$ $P2 = 0.001$ $P3 = 0.002$
	Yes	1 (2.94)	26 (70.27)	17 (34.69)	
Nodules	No	32 (94.12)	27 (72.97)	47 (95.92)	$P = 0.002$ $P1 = 0.051$ $P2 = 0.999$ $P3 = 0.011$
	Yes	2 (5.88)	10 (27.03)	2 (4.08)	
Exophthalmos	No	34 (100.00)	11 (29.73)	47 (95.92)	$P < 0.001$ $P1 < 0.001$ $P2 = 0.51$ $P3 < 0.001$
	Yes		26 (70.27)	2 (4.08)	
FT3 (Pg⁄ml)	Median (IQR)	1.73 (1.45–1.99)	4.80 (4.38–5.73) †	0.43 (0.25–0.53) †*	$P < 0.001$ $P1 < 0.001$ $P2 < 0.001$ $P3 < 0.001$
FT4 (ng⁄dl)	Median (IQR)	12.91 (10.47–15.15)	28.53 (26.65–32.63) †	4.63 (3.28–5.72) †*	$P < 0.001$ $P1 < 0.001$ $P2 < 0.001$ $P3 < 0.001$
TSH (mlu⁄l)	Median (IQR)	1.74 (0.56–4.14)	0.03 (0.01–0.14) †	9.47 (8.53–12.76) †*	$P < 0.001$ $P1 = 0.001$ $P2 < 0.001$ $P3 < 0.001$
TGAb (Iu⁄ml)	Median (IQR)	62.62 (49.06–73.25)	182.98 (154.00–205.34) †	85.83 (65.00–151.50) †*	$P < 0.001$ $P1 < 0.001$ $P2 < 0.001$ $P3 < 0.001$
TRAB (mlu⁄ml)	Median (IQR)	1.20 (0.89–1.40)	3.23 (2.30–4.80) †	1.46 (1.20–1.90) †*	$P < 0.001$ $P1 < 0.001$ $P2 = 0.006$ $P3 < 0.001$
HBA1c (%)	Median (IQR)	5.30 (4.90–5.47)	6.20 (4.80–6.80) †	6.50 (4.90–7.50) †	$P = 0.004$ $P1 = 0.034$ $P2 = 0.003$ $P3 = 0.456$
FBS (mg⁄dl)	Median (IQR)	92.35 (84.25–110.00)	130.00 (100.75–140.00) †	127.00 (100.65–140.00) †	$P = 0.001$ $P1 = 0.001$ $P2 = 0.002$ $P3 = 0.643$
2Hr.pp (mg⁄dl)	Median (IQR)	154.50 (145.00–165.00)	185.00 (150.00–235.00) †	184.00 (155.00–228.00) †	$P = 0.019$ $P1 = 0.036$ $P2 = 0.036$ $P3 = 0.927$
HOMA	Median (IQR)	1.20 (0.83–1.50)	1.70 (1.20–2.20) †	1.60 (1.30–2.80) †	$P < 0.001$ $P1 = 0.004$ $P2 < 0.001$ $P3 = 0.439$

2hr.pp, 2 h post-prandial; FBS, fasting blood sugar; Ft3, free triiodothyronine; Ft4, free thyroxine; HbA1c, glycosylated hemoglobin; HOMA, homeostatatic model assessment for insulin resistance; IU/ml, international unit/milliliter; mg/dl, milligram/deciliter; mIu/ml, milli international unit/milliliter; mlu⁄l, milli international unit; n, number; ng/dl, nanogram/deciliter; *P*.value, probability value; pg/ml, picogram/milliliter; TGAb, thyroglobulin antibody; TRAB, thyroid stimulating hormone receptor antibody; TSH, thyroid stimulating hormone; P: comparison between the 3 group.

*P*1: comparison between hyperthyroid and normal group.

*P*2: comparison between hypothyroid and normal group.

*P*3: comparison between hyperthyroid and hypothyroid group.

P.value < 0.05 is significant, *P*.value < 0.001 is highly significant.

Numerical data are shown as median (IQR), analyzed by the Kruskal Wallis test followed by the Dunn test, other data are shown as count (percent), analyzed by a Chi-squared test. † Compared to the normal group, * compared to the hyperthyroid group.

Post hoc analysis of clinical characteristics by Dunn test/Fisher test.

Table 3. Correlation of thyroid and diabetic Laboratory tests.

	FT3	FT4	TSH	TGAb	TRAB
HBA1c (%)	−0.0492382 ($P = 0.593$)	−0.1188966 ($P = 0.159$)	0.0507236 ($P = 0.582$)	0.2094443 ($P = 0.021$)	0.0630131 ($P = 0.494$)
FBG (mg\dl)	0.0174082 ($P = 0.85$)	−0.0816244 ($P = 0.375$)	−0.0314711 ($P = 0.732$)	0.1349220 ($P = 0.141$)	0.0994276 ($P = 0.279$)
X2Hr.pp (mg\dl)	0.0287585 ($P = 0.755$)	−0.0769061 ($P = 0.404$)	−0.0157129 ($P = 0.864$)	0.0810745 ($P = 0.378$)	0.0662382 ($P = 0.472$)
HOMA	−0.0670653 ($P = 0.466$)	−0.1392744 ($P = 0.192$)	0.1054446 ($P = 0.251$)	0.2023754 ($P = 0.026$)	0.1838697 ($P = 0.044$)

2 h.pp, 2 h post-prandial; FBG, fasting blood glucose; FT3, free triidothyronine; FT4, free thyroxine; HBA1c, glycosylated haemoglobin; HOMA, homeostatatic model assessment for insulin resistance; mg/dl, milligram/deciliter; p, probability value; TGAb, thyroglobulin antibody; TRAB, thyroid stimulating hormone receptor antibody; TSH, thyroid stimulating hormone.
P.value < 0.05 is significant, *P*.value < 0.001 is highly significant.

(2017), who illustrated a higher prevalence of thyroid dysfunction in older persons due to subdividing the groups based on age (<60 and ≥ 60 years old). In agreement with World Health Statistics 2021, being with T2DM does not predict the possibility of hyperthyroid disease in the elder people with DM [6,7].

The current study, we found that increased HbA1c level by 0.017% for each kilogram increase in weight ($P < 0.05$), 0.063% for each kg/m^2 increase in BMI ($P < 0.05$), this is agreed with **Gummesson *et al.*** [8] who found that there is 0.1% reduction in mean HbA1c with each kilogram of mean weight loss. However, **Gummesson *et al.*** [8] have found that BMI alone doesn't predict type 2 diabetes due to differences in fat mass (especially abnormal adiposity) and muscle mass for the same BMI value.

The present study found that increased HbA1c level by 0.024% for each mmHg elevation in systolic blood pressure ($P < 0.05$), 0.036% for each mmHg elevation in diastolic blood pressure ($P < 0.05$), in the same line **Petrie *et al.*,** [9] indicated that individuals with hypertension have a higher risk of developing diabetes than those with normal blood pressure.

Also, in this study we found thyroid dysfunction more in female (76 case) than male (10 cases), this agrees with **Biondi and Kahaly,** [10] who found that thyroid dysfunction was more predominant in females than males with a ratio of nearly 2:1.

In this study increased HbA1c level in hypothyroid patients by 1.05% ($P < 0.05$) compared to normal persons, similar results were observed by **Kumar *et al.*,** [11] who reported that statistically significant correlation by persons correlation coefficient was

Table 4. Multiple Linear regression model results for predictors of hemoglobin A1c level.

Dependent: HBA1c	Coefficient	95% CI	*P* value
Sex, F	0.166	−0.26 to 0.59	0.436
Age (years)	0.002	−0.01 to 0.02	0.803
Weight (kg)	0.005	−0.02 to 0.03	0.696
BMI (kg\m2)	−0.009	−0.08 to 0.06	0.795
FT3 (Pg\ml)	0.03	−0.02 to 0.08	0.263
FT4 (ng\dl)	−0.002	−0.04 to 0.04	0.923
TSH (mlu\l)	0.005	−0.01 to 0.02	0.434
TGAb (Iu\ml)	0.002	0.00 to 0.003	0.016
TRAB (mlu\ml)	−0.021	−0.04 to −0.01	0.008
FBS (mg\dl)	0.013	0.003 to 0.02	0.011
2Hr.pp (mg\dl)	0.013	0.01 to 0.02	<0.001
HOMA	0.13	−0.04 to 0.30	0.134
Disease	–		–
Hyperthyroidism	−0.32	−1.04 to 0.39	0.371
Hypothyroidism	0.12	−0.41 to 0.65	0.660

2 h.pp, 2 h post-prandial; F, female; FBG, fasting blood glucose; FT3, free triiodothyronine; FT4, free thyroxine; HBA1c, glycosylated haemoglobin; HOMA, homeostatatic model assessment for insulin resistance; IU/ml, international unit/milliliter; kg/m^2, kilogram/meter^2; Kg, kilogram; mg/dl, milligram/deciliter; mIu/ml, milli international unit/milliliter; mlu\l, milli international unit; ng/dl, nanogram/deciliter; *P*. value, probability value; pg/ml, picogram/milliliter; TGAb, thyroglobulin antibody; TRAB, thyroid stimulating hormone receptor antibody; TSH, thyroid stimulating hormone;
P.value < 0.05 is significant, *P*.value < 0.001 is highly significant.

Table 5. Multiple logistic regression model results for predictors of diabetes

Dependent:	Odds ratio	95% CI	*P* value
Sex, F	3.05	0.76−15.22	0.136
Age (Years)	1.03	0.99−1.08	0.088
Weight (kg)	1.00	0.92−1.09	0.966
BMI (kg\m2)	1.12	0.89−1.39	0.324
FT3 (Pg\ml)	1.07	0.90−1.29	0.424
FT4 (ng\dl)	0.86	0.72−0.98	0.043
TSH (mlu\l)	0.97	0.92−1.01	0.223
TGAb (Iu\ml)	1.00	0.99−1.01	0.782
TRAB (mlu\ml)	1.03	0.98−1.10	0.267
Disease, Normal	–	–	–
Hyperthyroidism	55.63	5.27−1003.71	0.003
Hypothyroidism	0.90	0.14−5.42	0.911

(FT3: free triiodothyronine, FT4: free thyroxine, TSH: thyroid stimulating hormone, TGAb: thyroglobulin antibody, TRAB: thyroid stimulating hormone receptor antibody, *P*.value: probability value, Kg: kilogram, kg/m^2: kilogram/meter^2, pg/ml: picogram/milliliter, ng/dl: nanogram/deciliter, mlu\l: milli international unit, IU/ml: international unit/milliliter, mg/dl: milligram/deciliter, mIu/ml: milli international unit/milliliter.
P.value < 0.05 is significant, *P*.value < 0.001 is highly significant.

Table 6. Risk of diabetes mellitus between normal and thyroid diseases

	Not diabetic	Diabetic	*P* value	RR (CI: 95%)
Normal	28 (82.4)	6 (17.6)		Reference
Hyperthyroidism	15 (40.5)	22 (59.5)	<0.001	3.36 (1.55−7.30)
Hypothyroidism	22 (44.9)	27 (55.1)	<0.001	3.12 (1.44−6.74)

P.Value: probability value.
P.value < 0.05 is significant, *P*.value < 0.001 is highly significant.

found between the serum levels of HbA1c and TSH in patients with hypothyroidism [12].

In our study increased HBA1c level in hyperthyroid patients by 0.76%. In contrast (**Biondi and Cooper** [13]), found that hyperthyroid patients had the same mean HbA1c value as controls at the baseline.

As regard the correlations between thyroid autoantibodies levels or autoimmune thyroid disease and DM, we found that decreased HbA1c level by 0.021% for each 1 mlu⁄ml increase in TRAB. Previous studies found that autoimmune thyroid disease or thyroid autoantibodies levels were not significantly related with DM, in patients with T2DM and with type 1 DM as well [14].

This study showed that decreased odds of being a diabetic with increased free T3 (OR: 0.86, $P < 0.05$), increased odds of being a diabetic with hyperthyroid patients compared to normal (OR: 55.63, $P < 0.05$). On other hand, **Blanc *et al.***, found the effect of control of diabetes on the level of thyroid hormones, they found that a poor diabetes control (HbA1c $\geq$ 12%) predisposes to low T3 syndrome due to impairment of T4 to T3 conversion [15].

In our study we found that hyperthyroid patients have 3.36 times the risk of being diabetic compared to normal. In accordance with **Fleiner HF *et al.***, [16] who reported that hyperthyroid disease leads to hypermetabolic state with more weight loss and energy expenditure in spite of increased food intake and appetite, increased gluconeogenesis and lipolysis. Euthyroidism can improve insulin resistance associated with hyperthyroid disease.

This study showed that, Hypothyroid patients have 3.12 times the risk of being diabetic compared to normal. In agreement with **Gronich *et al.***, [17] who reported a high risk of DM in patients with hypothyroidism. Moreover, **Smith EA *et al.***, [18] found that there was significant correlation between HbA1c and TSH levels. Our study suggests that we should be careful while analyzing HbA1c data in patients with hypothyroid disease.

4.1. Conclusion

Blood glucose levels should be done in patients after the initial diagnosis of thyroid diseases for early diagnosis and a good treatment of DM.

Re-evaluation of the status of blood glucose and considering the need for long-term antidiabetic treatment is necessary once thyroid function restore to normal.

Conflicts of interest

There are no conflicts of interest.

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