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ORIGINAL STUDY

Predictors of obesity in hypothyroid patients under treatment

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Abstract

Objectives: **To evaluate obesity and insulin resistance relation in treated hypothyroid patients attending Menoufia University Hospital.**

Background: **Hypothyroidism is a common endocrine disorder resulting from deficient secretion of thyroid hormone. Africa has the highest prevalence (14.2%) while Asia has the lowest prevalence (5.8%). 'Obesity' is a disease where body fat and body fat distribution exceeds the level considered healthy. Through the production of adipokines, adipose tissue affects the activity of the Hypothalamic−pituitary−thyroid (HPT) axis system. Insulin resistance and autoimmune thyroid disease have been shown to influence each other.**

Patient and methods: **Our study included 254 Egyptian hypothyroid patients under treatment divided into two groups group A (hypothyroid obese) and group B (hypothyroid nonobese) to study Obesity predictors in treated hypothyroid patients attending Menoufia University Hospital. They were assessed for systolic blood pressure, diastolic blood pressure, BMI and investigated for Serum levels of thyroid-stimulating hormone, serum levels of free triiodothyronine (FT3), free thyroxine (FT4), fasting blood glucose (FBG), homeostasis model assessment of insulin resistance, Thyroid peroxidase (Anti-TPO) antibody, fasting serum lipid profile and total leucocytic counts (neutrophils and lymphocytes).**

Results: **Homeostasis model assessment of insulin resistance, systolic blood pressure, Total triglycerides, diastolic blood pressure, and FT4 was ranked in descending order by odd ratio as independent predictors of obesity, with the exception of free triiodothyronine, fasting blood glucose, total cholesterol, and low-density lipoprotein cholesterol.**

Conclusion: **Targeting multiple factors (insulin resistance, good control of blood pressure, treatment of hypertriglyceridemia, and control of low FT4) is a priority to lose weight and get rid of obesity in hypothyroid patients.**

Keywords: Hypertriglyceridemia, Hypothyroidism, Insulin resistance, Obesity

1. Introduction

Obesity is accompanied by various physiological and pathological changes in the human's body, including the thyroid gland. The prevalence of severe obesity will increase by 130% in 2030. It has become a worldwide health problem [1].

Many studies have investigated the relationship between obesity and thyroid dysfunction, but the results are still inconsistent [2].

Raised BMI is a major risk factor for diseases as cardiovascular diseases (mainly heart disease and stroke), diabetes under treatment, musculoskeletal disorders (especially osteoarthritis), and cancers (endometrial, breast, ovarian, prostate, etc) [3].

When looking for obesity causes, thyroid hormonal tests are usually ordered. Typically, thyroid-stimulating hormone (TSH) levels are within the reference range or slightly higher in the obese state. The relationship between the hypothalamic−pituitary−thyroid axis (HPT) and obesity is complex and involves multiple interactions [4].

Insulin resistance is primarily an acquired condition related to excess body fat, although genetic causes are also identified [5].

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Hyperglycemia, hypertension, dyslipidemia, hyperuricemia, high inflammatory markers, endothelial dysfunction, and a prothrombotic state are the metabolic consequences of insulin resistance that can progress [6].

The Homeostatic Model Assessment (HOMA) method has been used by most studies on insulin resistance and thyroid diseases to evaluate individuals' insulin resistance [7].

2. Patients and methods

A cross-sectional study assessed 254 hypothyroid Egyptian patients under treatment. They were divided into two groups, group A (hypothyroid obese) and group B (hypothyroid nonobese) to study obesity predictors in treated hypothyroid patients attending outpatient clinics in Menoufia University Hospital. Samples were collected according to inclusion and exclusion criteria from December 2021 to December 2022, written informed consent was obtained from all participants and the study was approved by the Research Ethical Committee of Menoufia University.

Our study excluded patients with complex cardiovascular diseases, liver or kidney diseases, inflammatory bowel diseases, and rheumatoid diseases, as well as those with pregnancy, lactation, diabetic treatment, cortisol therapy, Cushing syndrome, and PCOs.

All patients were subjected to thorough history taking of age, sex, occupation, smoking, type of salt intake, activity, prior disease history, and use of medications. Height and body weight of all participants were measured. BMI is determined as weight in kilograms divided by the square of height in meters (kg/m^2) [3].

BMI Level	Weight classification
Below 18.5	Underweight
18.5 to <25	Healthy weight
25 to <30	Overweight
30 and above	Obesity
40 and above	Severe obesity

MI levels for adults ages 20 and over [3].

Serum levels of TSH of all patients by (mIU/L), free triiodothyronine (FT3) and free thyroxine (FT4) were measured with an electrochemiluminescence immunoassay, fasting blood glucose (FBG), homeostasis model assessment of insulin resistance (HOMA-IR), HOMA-IR was calculated according to the following formula: FBG (mg/dL) × fasting insulin (mU/mL)/405 [8]. Patients with HOMA-IR scores higher than 1.8 were accepted to have IR. Thyroid peroxidase (Anti-TPO) antibody levels were measured with an electrochemiluminescence immunoassay on a Cobas 601 analyzer. Lipid profile measured by a fasting lipid panel with an automatic biochemical analyzer (Mindray BS-180 Analyzer) and total leucocytic counts (neutrophils and lymphocytes).

Blood for hormonal and biochemical analyses was collected after an overnight fasting in serum tubes between 7.30 and 10.00 a.m. The samples were routinely processed and analyzed.

2.1. Statistical analysis

Statistical analysis was performed using SPSS (Statistical Package for the Social Sciences) version 26 (IBM Inc., Armonk, NY, USA). Quantitative data distribution was analyzed with the Shapiro-Wilks normality test and histograms to determine whether parametric or nonparametric statistical testing was warranted. The *F* test was used to compare the three groups, and the Tukey *post hoc* test was used to compare the means and SD of each pair of groups for any parametric variables (such as age) that were reported. The paired *T*-test was used to evaluate the relationship between two continuous variables collected from the same set of patients. Categorical variables (like sex) were reported as frequencies and percentages and analyzed statistically using the χ^2 test. In other words, r is the linear correlation coefficient. One group's correlation between two quantitative variables was calculated. Diagnosis performance is assessed via sensitivity, specificity, positive predictive value, and negative predictive value. The factors that were found to be unrelated to the development of the disease were assessed using univariate regression. Statistical significance was assumed when the two-tailed *P* value was less than or equal to 0.05.

3. Results

In our study males were 12.2% (31) of the hypothyroid patients under treatment and 87.8% (223) were females hypothyroid.

Our results in Table 1 showed the Mean age (39.25 ± 7.32) years, mean weight (83.32 ± 17.81) kg, mean height (163.07 ± 6.30) cm, mean BMI (31.30 ± 6.06), mean systolic blood pressure (SBP) (108.66 ± 15.47) mmHg, mean of diastolic blood pressure (DBP) (70.24 ± 9.94) mmHg, percent of smokers was 7.1% and nonsmokers was 92.9%, patients with absent medication history were 173 (Table 1).

TSH median was 3.03 (1.79–6.1) mIu/L, FT3 (2.56 ± 0.83) mIu/L, FT4 (1.22 ± 0.25) mIu/L, Anti

Table 1. Sociodemographic data of hypothyroid patients (n = 254).

Variable	Number of studied patients = 254 *n* (%)
Sex	
Male	31 (12.2)
Female	223 (87.8)
Age (years)	
Mean ± SD	39.25 ± 7.32
Range	21–57
Weight (Kg)	
Mean ± SD	83.32 ± 17.81
Range	40–120
Height (cm)	
Mean ± SD	163.07 ± 6.30
Range	150–178
BMI (Kg/m^2)	
Mean ± SD	31.30 ± 6.06
Range	22–46
SBP (mmHg)	
Mean ± SD	108.66 ± 15.47
Range	90–150
DBP (mmHg)	
Mean ± SD	70.24 ± 9.94
Range	60–90
Smoking	
Smoker	18 (7.1)
Nonsmoker	236 (92.9)
Medication history	
Absent	173 (68.1)
Analgesic	28 (11)
Antihypertension	23 (9.1)
Statins	4 (1.6)
Vitamins	24 (9.4)
Orilistat	2 (0.8)
Salt intake	
Low	204 (80.3)
Medium	50 (19.7)
Activity	
Average	187 (73.6)
High	67 (26.4)
Prior diseases	
Absent	115 (45.3)
Diet control	99 (39)
Hypertension	21 (8.3)
Hypotension	13 (5.1)
Migraine	4 (1.6)
Anaemia	1 (0.4)
Viral infection	1 (0.4)

%, hundred percent; BMI, Body mass index; Cm, centimetre; DBP, Diastolic blood pressure; Kg, kilogram; m^2, meter square; mmHg, millimetre mercury; NO, number; SBP, Systolic blood pressure; SD, Standard deviation.

TPO 12 (1.04–330) IU/mL, FBG (89.76 ± 9.98) mg/dL, HOMA IR 2.3 (1.2–3)U, total triglycerides (TGs) (101.74 ± 32.72) mg/dL, total cholesterol (192.22 ± 43.99) mg/dL, low-density lipoprotein cholesterol (LDL-C) (107.34 ± 29.92) mg/dl, high-density lipoprotein cholesterol (HDL-C) (47.67 ± 12.53) mg/dL, white blood cells (WBCs) 7.8 (6.7–8.7) × 10^3 u/l, Neutrophils 4.1 (3.45–4.5) × 10^3 u/l, and lymphocytes 2 (1.7–2.5) × 10^3 u/l (Table 2).

Table 2. Laboratory findings in hypothyroid patients under treatment (n = 254).

Variable	Number of studied patients = 254
TSH (mIu/l)	
Median (IQR)	3.03 (1.79–6.1)
Range	0.3–30
FT3 (mIu/l)	
Mean ± SD	2.56 ± 0.83
Range	0.9–4
FT4 (mIu/l)	
Mean ± SD	1.22 ± 0.25
Range	0.82–2
Anti TPO (IU/mL)	
Median (IQR)	12 (1.04–330)
Range	0.1–7521
FBG (mg/dl)	
Mean ± SD	89.76 ± 9.98
Range	70–114
HOMAIR (U)	
Median (IQR)	2.3 (1.2–3)
Range	0.55–8.87
TG (mg/dl)	
Mean ± SD	101.74 ± 32.72
Range	58–280
TC (mg/dl)	
Mean ± SD	192.22 ± 43.99
Range	115–295
LDL-C (mg/dl)	
Mean ± SD	107.34 ± 29.92
Range	52–212
HDL-C (mg/dL)	
Mean ± SD	47.67 ± 12.53
Range	28–110
WBCs (×10^3 u/l)	
Median (IQR)	7.8 (6.7–8.7)
Range	4.5–107
Neutrophils (×10^3 u/l)	
Median (IQR)	4.1 (3.45–4.5)
Range	1.06–61
Lymphocytes (×10^3 u/l)	
Median (IQR)	2 (1.7–2.5)
Range	1–4.5

Anti TPO, Thyroid peroxidase antibody levels; FBG, Fasting blood glucose; FT3, Free triiodothyronine; FT4, Free thyroxine; HDL-C, High-density lipoprotein cholesterol; IQR, Interquartile range; LDL-C, Low-density lipoprotein cholesterol; mg/Dl, milligram per decilitre; mIu/L, Mille international unit per litre; SD, Standard deviation; TC, Total cholesterol; TG, Total triglycerides; TSH, Thyroid-stimulating hormone; unit, IU, international unit; WBCs, White blood corpuscles.

A significant difference in SBP, DBP, weight, and BMI appeared between obese (149) patients and nonobese (105) hypothyroid patients (Table 3).

There was a significant difference between both groups according to weight where it was higher in obese than nonobese hypothyroid patients. BMI showed a significant difference between both groups. SBP was higher in obese than nonobese with a significant difference. Another significant difference between both groups appeared in DBP (Fig. 1).

Table 3. Comparison between obese and nonobese patients regarding their sociodemographic data (n = 254).

Variable	Obese ($N = 149$) [n (%)]	Nonobese ($N = 105$) [n (%)]	Test of significance	P value
Sex				
Male	15 (10.1)	16 (15.2)	$\chi^2 = 1.54$	0.215 (NS)
Female	134 (89.9)	89 (84.8)		
Age (years)				
Mean ± SD	39.43 ± 7.14	39.10 ± 7.37	t = 0.70	0.484 (NS)
Range	21–56	21–57		
Weight (Kg)				
Mean ± SD	94.19 ± 15.59	68.14 ± 5.33	$t = 16.42$	**<0.001**[a]
Range	40–120	57–80		
Height (cm)				
Mean ± SD	163.55 ± 7.02	162.54 ± 5.10	t = 1.30	0.195 (NS)
Range	150–178	156–173		
BMI (Kg/m^2)				
Mean ± SD	35.63 ± 4.01	25.19 ± 1.68	t = 24.97	**<0.001**[a]
Range	30–46	22–28		
SBP (mmHg)				
Mean ± SD	113.61 ± 16.76	101.46 ± 9.44	t = 6.58	**<0.001**[a]
Range	90–150	90–140		
DBP (mmHg)				
Mean ± SD	72.38 ± 11.43	66.99 ± 6.08	t = 4.17	**<0.001**[a]
Range	60–90	60–80		
Smoking				
Smoker	8 (5.4)	10 (9.5)	$\chi^2 = 1.62$	0.204 (NS)
Nonsmoker	141 (94.6)	95 (90.5)		
Medication history				
Present	50 (33.6)	31 (29.5)	$\chi^2 = 0.46$	0.497 (NS)
Absent	99 (66.4)	74 (70.5)		
Salt intake				
Low	123 (82.6)	81 (77.1)	$\chi^2 = 1.14$	0.286 (NS)
Medium	26 (17.4)	24 (22.9)		
Activity				
Average	114 (76.5)	73 (69.5)	$\chi^2 = 1.55$	0.213 (NS)
High	35 (23.5)	32 (30.5)		
Prior diseases				
Present	82 (55)	57 (54.3)	$\chi^2 = 0.01$	0.906 (NS)
Absent	67 (45)	48 (45.7)		

[a] Statistically significant.

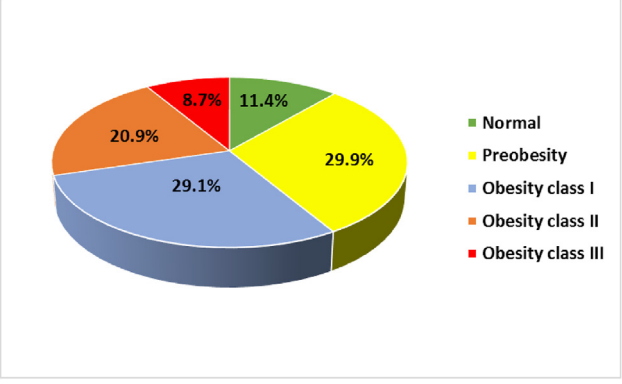

Fig. 1. Prevalence of obesity in hypothyroid studied patients (n = 254).

A significant difference in FT3 between obese (2.40 ± 0.80) mIu/L and nonobese (2.81 ± 0.81) mIu/L a significant difference also regarding FT4 between obese 1.15 ± 0.22 and nonobese (1.34 ± 0.25) mIu/L a significant difference appeared in FBS where higher in obese is (92.67 ± 10.38) mg/dl than (85.48 ± 7.53) mg/dl, another significant difference is seen in HOMA IR where it was higher in obese (85.48 ± 7.53U) than nonobese (1.81 ± 1.09 U), also a significant difference in TGs between both groups as the mean in obese was 110.32 ± 39.02 mg/dl and in nonobese was 89.55 ± 13.76 mg/dl, total cholesterol (TC) shows a significant difference between obese (201.13 ± 39.35) and nonobese (179.34 ± 47.36) mg/dl. Another significant difference in LDL-C appears between obese (114.07 ± 26.73) mg/dl, and nonobese (97.30 ± 31.37) mg/dl, which were higher in obese than nonobese hypothyroid subjects. Results showed a nonsignificant difference between both obese and nonobese in TSH, Anti TPO, HDL-C, WBCS, neutrophils, and lymphocytes (Table 4).

Binary logistic regression analysis was run to ascertain the effects of systolic blood pressure,

Table 4. Comparison between obese and nonobese patients regarding their laboratory data (n = 254).

Variable	Obese $n = 149$	Nonobese $n = 105$	Test of significance	P value
TSH (mIu/l)				
Mean ± SD	4.60 ± 4.81	4.53 ± 4.81	U = 1.95	0.051 (NS)
Range	0.79–27	0.3–30		
FT3 (mIu/l)				
Mean ± SD	2.40 ± 0.80	2.81 ± 0.81	t = 3.68	**<0.001**[a]
Range	1–4	0.9–4		
FT4 (mIu/l)				
Mean ± SD	1.15 ± 0.22	1.34 ± 0.25	t = 6.23	**<0.001**[a]
Range	0.82–2	0.96–1.8		
Anti TPO (IU/ml)				
Mean ± SD	560.96 ± 1381.67	247.97 ± 440.99	U = 0.92	0.358 (NS)
Range	0.1–7521	0.1–1520		
FBG (mg/dl)				
Mean ± SD	92.67 ± 10.38	85.48 ± 7.53	t = 6.05	**<0.001**[a]
Range	70–114	76–100		
HOMA IR (U)				
Mean ± SD	2.99 ± 1.70	1.81 ± 1.09	U = 6.42	**<0.001**[a]
Range	0.92–8.87	0.55–4.2		
TGs (mg/dl)				
Mean ± SD	110.32 ± 39.02	89.55 ± 13.76	U = 5.70	**<0.001**[a]
Range	58–280	68–155		
TC (mg/dl)				
Mean ± SD	201.13 ± 39.35	179.34 ± 47.36	t = 3.36	**0.001**[a]
Range	120–280	115–295		
LDL-C (mg/dl)				
Mean ± SD	114.07 ± 26.73	97.30 ± 31.37	t = 4.11	**<0.001**[a]
Range	52–190	60–212		
HDL-C (mg/dL)				
Mean ± SD	47.10 ± 11.65	48.57 ± 13.70	t = 0.91	0.362 (NS)
Range	28–88	38–110		
WBCs ($\times 10^3$ u/l)				
Mean ± SD	8.11 ± 1.59	11.09 ± 17.05	U = 1.79	0.07 (NS)
Range	5.3–14.5	4.5–107		
Neutrophils ($\times 10^3$ u/l)				
Mean ± SD	4.80 ± 6.68	4.53 ± 2.30	U = 0.21	0.833 (NS)
Range	1.06–61	1.07–14.1		
Lymphocytes ($\times 10^3$ u/l)				
Mean ± SD	2.23 ± 0.80	2.12 ± 0.76	U = 1.63	0.103 (NS)
Range	1.2–4.5	1–3.9		

Anti TPO, Thyroid peroxidase antibody levels; FBG, Fasting blood glucose; FT3, Free triiodothyronine; FT4, Free thyroxine; HDL-C, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; NS, Non-significant; SD, Standard deviation; t, Student *t*-test; TG, Total triglycerides, TC:Total cholesterol; TSH, Thyroid-stimulating hormone; U, Mann-Whitney *U* test; WBCs, White blood cells.

[a] Statistically significant.

diastolic blood pressure, FT3, FT4, FBG, HOMA IR, TG, TC, and LDL-C on the likelihood of development of obesity in all hypothyroid participants. On univariable analysis, all predictor variables were statistically significant. Accordingly, they entered into a regression model. Of predictor variables, all were statistically significant independent predictors of obesity except FT3, FBG, TC, and LDL-C. The order of factors according to their odd's ratio was HOMA-IR 2.826 [95% confidence interval (CI) 1.070–1.190, P value < 0.001*], SBP 1.128 (95% CI 1.070–1.190, P value < 0.001*), TGs (95% CI 1.013–1.046, P value < 0.001*), DBP 0.887 (95% CI 0.824–0.955, P value 0.002*) and FT4 0.046 (95% CI 0.011–0.199, P value < 0.001*) in a descending manner (Table 5).

4. Discussion

Our results showed that the mean age in the obese group was 39.43 ± 7.14 years and nonobese was 39.10 ± 7.37 years which shows no significant difference between both groups. Our result is closely near to the finding of Choi *et al.* [9], the mean age of their hypothyroid subjects was 37.99 ± 0.23 years.

Our finding showed that females had the highest ratio in hypothyroid patients as the female number is 223 (87.8%) patients higher than the male 31

Table 5. Predictors of obesity among the 254 hypothyroid studied patients.

	Univariable			Multivariable		
Predictor	cOR	95% CI	*P* value	aOR	95% CI	*P* value
HOMA IR (U)	2.090	1.596–2.737	<0.001[a]	2.826	1.859–4.296	<0.001[a]
SBP (mmHg)	1.070	1.044–1.096	<0.001[a]	1.128	1.070–1.190	<0.001[a]
TG (mg/dL)	1.041	1.021–1.060	<0.001[a]	1.029	1.013–1.046	<0.001[a]
DBP (mmHg)	1.058	1.029–1.088	<0.001[a]	0.887	0.824–0.955	0.002[a]
FT4 (mIu/L)	0.036	0.011–0.122	<0.001[a]	0.046	0.011–0.199	<0.001[a]
FT3 (mIu/L)	0.562	0.409–0.772	<0.001[a]	0.596	0.353–1.004	0.052
FBG (mg/dL)	1.085	1.053–1.118	<0.001[a]	1.028	0.984–1.074	0.215
TC (mg/dL)	1.011	1.004–1.017	0.001[a]	0.986	0.973–1.000	0.052
LDL-C (mg/dL)	1.021	1.010–1.031	<0.001[a]	1.012	0.991–1.033	0.277

aOR, Adjusted odds ratio; CI, Confidence interval; cOR, Crude odds ratio.
[a] Statistically significant.

(12.2%) patients and the nonsmokers were 92.9% higher than smokers 7.1%.

These results came in agreement with Jiashu *et al.* [10], which stated that prevalence of hypothyroidism was significantly higher in women than in men (1.53% vs. 0.53%, $P < 0.001$). The same results were agreed with Mele *et al.* [11], study.

The prevalence of nonsmoking in hypothyroid patients was high but it did not differ between obese and nonobese. Our results mimic the study by Mele *et al.* [11], which showed that females and nonsmokers had higher TSH levels.

Our explanation for these results may be because of the higher prevalence of hypothyroidism in females and does not mean that smoking is a protective mechanism from hypothyroidism or Obesity.

The prevalence of obesity was higher in hypothyroid females (134, 89.9%) than in males (15 of 10.1%), 0.215, our results are inconsistent with Mahdavi *et al.* [2], who resulted that the prevalence of obesity was significantly higher among female participants than males.

Our study showed a significant difference in BMI in obese hypothyroid than nonobese hypothyroid patients under treatment which were parallel to Bernadette Biondi and Naoki Tanaka's [12] study on that stated high BMI was shown to considerably increase serum TSH levels; as a result, a rise in BMI can causally increase free T3. The same results were found in Bambini *et al.* study [13].

SBP in our research is the second factor as a predictor of obesity in hypothyroid patients (1.128, 95%) CI (1.070–1.190) this result was in agreement with Pluta *et al.* [14].

Our results showed a significant difference in DBP between obese (72.38 ± 11.43) mmHg and nonobese patients (66.99 ± 6.08) mmHg, *P* value less than 0.001*, confirmed by the regression analysis as DBP odd's ratio was 0.887 (95% CI 0.824–0.955) *P* value 0.002*. Our results were in agreement with Anupam *et al.* [15], who stated that overt hypothyroidism is associated with diastolic hypertension, reduced vasodilation, and increased arterial stiffness.

The results of our study showed a nonsignificant difference in TSH between both obese (4.60 ± 4.81) and nonobese (4.53 ± 4.81), *P* value more than 0.05. TSH was nonsignificant predictor of obesity in hypothyroid patients, this came in contrast to Guo *et al.* [16], who found that TSH rose 2.201 times in the obese group.

This also came in contrast to Eduardo *et al.* [17], which cited that a positive correlation was found between TSH levels and obesity.

On the other hand, our results showed a significant difference in FT3 between obese (2.40 ± 0.80) and nonobese (2.81 ± 0.81) with *P* value less than 0.001. Our logistic regression supported this result as it appears that there is a negative association between higher levels of FT3 and the risk of becoming obese. This came in consistent with Bjergved *et al.* [18], a study proposed that obesity affects FT3, FT4, and causes high TSH with rising body weight and obesity.

Our results reported that FT4 had an important relationship with BMI and obesity as we found that FT4 in obese (1.15 ± 0.22) mIu/L was lower than nonobese (1.34 ± 0.25) mIu/L, *P* value less than 0.001*.

Our results were in contrast to the study of Eduardo *et al.* [17], who stated that no correlation was found in the statistical analysis between both Free T3 and Free T4 with obesity.

This finding is further supported by the fact that the alterations in thyroid function typically normalize following weight loss achieved through bariatric surgery [19].

Our results also came in contrast to Song *et al.* [20], study, which demonstrated that there was a negative correlation between FT4 and BMI and a positive relationship between TSH and BMI.

Our result showed a nonsignificant correlation between obesity and anti-TPO in obese hypothyroid

subjects (560.96 ± 1381.67) IU/ml and nonobese (247.97 ± 440.99) IU/ml, *P* value 0.358, our results were in contrast to Mahdavi *et al.* [2], who found that obesity had a positive correlation with TSH and anti-TPO, also came in contrast to Song *et al.* [21].

This result is consistent with Eduardo *et al.* [17], study where thyroid antibodies do not differ in obese patients and nonobese population.

Results of our study showed that FBG in obese patients (92.67 ± 10.38) mg/dl were higher than in nonobese (85.48 ± 7.53) mg/dl, *P* value less than 0.001*. The regression analysis showed no significant relation between FBG with obesity in hypothyroid patients. This could be interpreted by the fact that the thyroid hormone affects glucose homeostasis by impacting pancreatic β-cell development and glucose metabolism [22].

These results came in agreement with Zhang *et al.* [23] study which stated that nonsignificant higher fasting blood glucose in obese than nonobese people. The same results by Neelam *et al.* [24], which showed a positive correlation between fasting blood glucose level and BMI.

Our study showed a significant difference in HOMA-IR higher in obese (2.99 ± 1.70 U) than nonobese (1.81 ± 1.09 U), this is supported by the Binary logistic regression analysis which postulated that HOMA-IR was statistically the first significant independent factor with the highest odd's ratio 2.826 (95% CI 1.859–4.296, *P* value < 0.001*) in this descending order (HOMA-IR, SBP, TGs, DBP and FT4).

Although previous studies have suggested a close correlation between insulin resistance and obesity and thyroid diseases, we think we are the first to reach a clue and ordering the factors predictable to obesity in hypothyroid patients in an obvious descending manner like this.

Our results came consistent with the results of Choi *et al.* [9], study which showed that there was a significant association between IR and thyroid hormones.

Our results showed a significant difference in TGs between both obese (110.32 ± 39.02) mg/dl and nonobese (89.55 ± 13.76) mg/dl, it showed that TGs were the only statistically significant independent predictor of obesity of the lipid profile and the third significant independent factor of obesity in hypothyroid patients under treatment with odd's ratio 1.029 (95% CI 1.013–1.046, *P* value < 0.001*).

Our results augmented the importance of targeting high levels of triglycerides as a third factor to help hypothyroid obese patients lose weight.

Our results showed that TC showed a significant difference between obese (201.13 ± 39.35) mg/dl and nonobese (179.34 ± 47.36) mg/dl. Another significant difference in LDL-C appears between obese (114.07 ± 26.73) mg/dl higher than nonobese (97.30 ± 31.37) mg/dl. Other results showed that there was no significant difference between both groups regarding HDL-C levels. However, in the regression analysis of lipid items, TC and LDL-C were not statistically significant independent predictors of obesity.

Our results are in agreement with Huixing and Daoquan [25], who stated that low levels of FT3 and FT4 or high TSH in hypothyroidism could increase LDL-C and TGs. However, HDL function is impaired in hypothyroidism. The same results were found in Sinha *et al.* [26], study.

D'Ambrosio *et al.* [27], had explained that hypothyroidism reduces hepatic lipase activity, and leads to hypertriglyceridemia.

These results are against NICE guidelines, (2023) [28] which reported treating comorbidities and secondary causes of dyslipidemia like hypothyroidism before starting statins as they considered that hypothyroidism treatment with thyroxine replacement will improve hypertriglyceridemia without initial treatment of it.

In addition, our results were in contrast to Chen *et al.* [29], which did not find significant associations of TC, TG, HDL-C, and LDL-C with thyroid hormones or TSH.

Our results showed no significant difference between both groups according to WBCs, Neutrophils and Lymphocytes and nonsignificant *P* values less than 0.001*. Our explanation for these results is that inflammation in the thyroid does not correlate to obesity management.

Our results came in agreement to the results of Chen *et al.* [29], study that showed significant associations between adipokines, thyroid function, and HOMA-IR, only a positive association with FT3 in the hypothyroid group was observed.

Additionally, most studies only explored the association between obesity and thyroid disorders and barely investigated whether thyroid dysfunction is the cause or consequence of obesity.

Our study focuses on the relationship between obesity and hypothyroidism, which make our research considered a new and important study of hypothyroidism and obesity. This came in agreement with a large cohort study by Wang *et al.* [30], also showed a high correlation between obesity, metabolic abnormalities, and hypothyroidism, studies were done on TSH, BMI, and obesity.

4.1. Conclusion

In conclusion, our study evaluating the hypothyroid Egyptian population under treatment demonstrates that many factors increase the probability of obesity in hypothyroid patients even with treatment. Therefore, thyroid functions alone are acquitted of the accusation as a cause of obesity. The regression order factors (HOMA IR, SBP, TGs, DBP, and FT4) were found to be the most important in treating obesity, and they include correction of Insulin Resistance, good blood pressure control, treatment of hypertriglyceridemia, and control of low FT4.

Authors contribution

The authors contributed in the manuscript equally.

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There are no funds.

Ethics information

The study was approved by the Research Ethical Committee of Menoufia University under code no. 1/2022INTIM4.

Institutional review board (IRB) approval number

2571987.

Conflicts of interest

There are no conflicts of interest.

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