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Research Article

CORRELATION BETWEEN THYROID FUNCTION AND OBESITY IN TYPE 2 DIABETES INDIVIDUALS

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ABSTRACT

Objectives: In the present study, it is aimed to investigate the prevalence of thyroid dysfunction and blood glucose parameters and their correlation in Type 2 diabetes mellitus (T2DM) obese patients.

Methods: The present study was a cross-sectional and observational study conducted for a period of 1 year January 2020–January 2022. The patient demographic data, age, and gender were collected from the medical records. After an overnight fast, venous blood samples were collected in the morning for laboratory tests. Electrochemiluminescence immunoassays were used to assess serum free T3 (FT3), FT4, and thyroid stimulating hormone (TSH). Fasting blood glucose, postprandial blood glucose, and random sugar levels were measured using enzymatic method. Body mass index (BMI) was calculated as weight (kg) divided by squared height (m).

Results: In the present study, the mean values of thyroid harmones T3, T4 between the obese and non-obese individuals showed that no much significant variations might be due to low sample size. However, the mean values of TSH values were significantly higher 8.7ng/dl for obese individuals than non-obese individuals. There was a significant positive correlation that was identified for thyroid parameters TSH between obese and non-obese T2DM patients. On the other hand, there was a significant influence of postprandial blood sugar, random blood sugar, hemoglobin A1C, and BMI in obese and non-obese T2DM patients and insignificant influence for thyroid parameters T3 and T4. However, the results also showed that there was a highly significant influence of THS levels in obese T2DM patients.

Conclusion: The findings of the present investigation showed a high correlation between thyroid function, mainly TSH levels, and Type 2 diabetes, particularly in obese persons.

Keywords: Diabetes, Obese, Thyroid, Body mass index, Non-obese.

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INTRODUCTION

Obesity or obesity-related disorders such as metabolic syndromes, hypertension, hyperglycemia, and dyslipidemia can be caused by thyroid dysfunction (TD). Obese individuals frequently experience slight changes in thyroid hormone levels. Hypothyroidism and obesity are two prevalent clinical disorders that have a strong relationship. With the global prevalence of obesity experiencing an unprecedented surge, the connection has gained increasing significance. Patients typically believe that thyroid disease is a secondary cause of obesity [1,2]. Current research has shown the connection between thyroid hormones and associated aspects of obesity and suggests that alterations in thyroid stimulating hormone (TSH) may be secondary to obesity. Hypothyroidism is traditionally cited as a contributor to weight gain through altered metabolic efficiency, water retention, and decreased lipolysis [3]. Central obesity, also known as central obesity, is the accumulation of fat in the midsection of obese people. Central obesity causes a wide range of metabolic thyroid disorders and impacts body weight in multiple ways. Case–control studies, epidemiological surveys, and the development of obesity and insulin resistance were all linked to higher TSH levels, independent of hypothyroidism. Although the exact cause of this association is yet unknown, it is likely caused by a compensatory reaction that is controlled by leptin and intended to prevent weight gain. The most prevalent endocrine problems are obesity, Type 2 diabetes mellitus (T2DM), and thyroid conditions, which frequently coexist in the same people. Patients with Type 2 DM (T2DM) are more likely to have TD, which is often described as an abnormal thyroid function test result, than people without diabetes. TD can have a negative impact on metabolic management. A reduction in the synthesis of insulin is possible if hypothyroidism, the primary cause of TD in diabetic patients, is present. Due to an increase in beta-cell mass and an increase in insulin clearance, hyperthyroidism causes beta-cell responsiveness to catecholamine or glucose to rise [4,5]. Moreover, both hypothyroidism and hyperthyroidism have the power to affect how insulin is metabolized and so cause insulin resistance. The endocrine function of TSH in controlling thyroid gland function is well understood. Thyroid hormones subsequently play an important role in energy metabolism and substrate oxidation, which helps to maintain body weight. It is commonly recognized that hypothyroidism is linked to a slower metabolic rate and decreased thermogenesis, while hyperthyroidism is linked to weight loss. The measurement of thyroid function in people with extreme obesity has produced contradictory results and alterations in thyroid function are controversially discussed in relation to obesity status. Measures of obesity were found to be favorably connected with serum TSH and negatively related to free thyroxine (FT4), despite the fact that thyroid function is often adequate in obese people [6,7]. Hence, the present study is aimed to investigate the prevalence of TD and blood glucose parameters and their correlation in T2DM obese patients.

METHODS

The present study was a cross-sectional and observational study conducted for a period of 1 year January 2020–January 2022. The patient demographic data, age, and gender were collected from the medical records. The inclusion criteria include all the Type 2 diabetes

individuals (obese and non-obese) with TDs. The exclusion criteria include that the patients underwent thyroid surgery, liver or kidney dysfunction patients, Type 1 diabetes patients, and patients with chronic complications (diabetic peripheral neuropathy (DPN), diabetic foot, etc.). The test population includes all the T2DM obese patients and control population was T2DM non-obese patients. The study sample size involves 75 obese T2DM patients and 75 non-obese T2DM patients. Body mass index (BMI) was calculated as weight (kg) divided by squared height (m). After an overnight fast, venous blood samples were collected in the morning for laboratory tests. Electrochemiluminescence immunoassays were used to assess serum free T3 (FT3), FT4, and TSH. Normal ranges were provided by kit producers as follows: TSH 0.4–5.0 μIU/mL, FT3 2.63–5.70 pmol/L, and FT4 9.01–19.05 pmol/L. Euthyroid was considered if thyroid hormone levels fall within reference range and TD was considered if thyroid hormones fall outside the reference range. The diagnostic categories for TD were as follows: Subclinical hypothyroidism (increased TSH values with normal FT4 levels); overt hypothyroidism (increased TSH values with decreased FT4 levels); subclinical hyperthyroidism (decreased TSH values with normal FT4 levels); overt hyperthyroidism (decreased TSH values with increased FT4 levels); and low T3 syndrome (decreased FT3 values only or decreased FT3, TSH, and/or FT4 levels).

Glucose is oxidized by the enzyme glucose oxidase to give D-gluconic acid and hydrogen peroxide. Hydrogen peroxide in presence of enzyme peroxidase oxidizes phenol which combines with 4-Aminoantipyrine to produce a red color quinoneimine dye. The intensity of the color developed is proportional to glucose concentration in the sample. Fasting blood glucose, postprandial blood glucose, and random sugar levels were measured using enzymatic method and the cutoff point of fasting blood sugar (FBS) 126 mg/dl[2], 2 h plasma glucose >200 mg/dl, random plasma glucose >200 mg/dl, and hemoglobin A1C (HbA1C) 6% was considered as diagnostic criterion for the diabetes.

RESULTS

Depend on the age and gender-wise distribution of cases, more cases were reported in the age group 41–50 years followed by 51–60 years. Comparatively, more number of females were reported than males specifically these two age groups (Fig. 1). Table 1 showed the results of paired Tukey's test results for blood glucose parameters and thyroid function parameters in T2DM obese and non-obese individuals. The FBS values compared for non-obese and obese individuals, the mean value was bit higher for non-obese individuals 137mg/dl whereas the postprandial blood sugar (PPBS) values were higher for obese individuals 183mg/dl than non-obese individuals. The random sugar levels were higher for obese than non-obese test population 116 mg/dl. The levels of HbA1C were higher in obese individuals 7.8% than non-obese individuals 5.2%. However, in the present study, the mean values of thyroid hormones T3 and T4 between the obese and non-obese individuals showed that no much significant variations might be due to low sample size. However, the mean values of TSH values were significantly higher 8.7ng/dl for obese individuals than non-obese individuals. By performing a Pearson's correlation for blood glucose parameters and thyroid function parameters in T2DM obese and non-obese individuals, the results showed insignificant negative correlation for thyroid parameters T3 and T4 between obese and non-obese T2DM patients but significant positive correlation was identified for thyroid parameters TSH between obese and non-obese T2DM patients. However, glucose parameters showed insignificant positive correlations (Table 1). The results of paired T-test analysis for blood glucose parameters and thyroid function parameters in T2DM obese and non-obese individuals showed that there was significant influence of PPBS, random blood sugar (RBS), HbA1C, and BMI in obese and non-obese T2DM patients and insignificant influence for thyroid parameters T3 and T4 (Table 2). However, the results also showed that there was a highly significant influence of THS levels in obese T2DM patients. From the Tables 2 and 3, the results showed that the mean values of BMI for obese individuals were significantly higher than the non-obese individuals. Hence, BMI was considered as a significant factor of obesity.

DISCUSSION

Obesity is a medical condition that occurs when there is too much body fat, and becomes a health risk factor. Obesity raises the risk of several illnesses and ailments, especially heart problems, Type 2 diabetes, obstructive sleep apnea, specific types of cancer, osteoarthritis, and depression. The most typical combination of factors leading to obesity is an excessive diet intake, a lack of physical activity, and genetic predisposition [7]. The thyroid gland's ability to function is impacted by the medical condition known as thyroid disease. It is an endocrine hormone since it is produced by the thyroid gland, which is situated at the front of the neck and helps regulate numerous other organs through the blood. These hormones normally act in the body to regulate energy use, infant development, and childhood development. Hypothyroidism and obesity are two prevalent clinical disorders that have a strong relationship [8]. With the global prevalence of obesity experiencing an unprecedented surge, the connection has gained increasing significance. Patients typically believe that thyroid disease is a secondary cause of obesity. A novel perspective suggests that alterations in thyroid-stimulating hormone (TSH) may be a result of obesity. Recent research has also shown a relationship between thyroid autoimmunity and obesity, with the adipocyte hormone leptin appearing to be the primary mediator between these two diseases. In this research, we were evaluating the intriguing connection between obesity and hypothyroidism, along with the ensuing clinical implications [9]. Reduced metabolic rate, decreased thermogenesis, a higher BMI, and a higher prevalence of obesity have all been

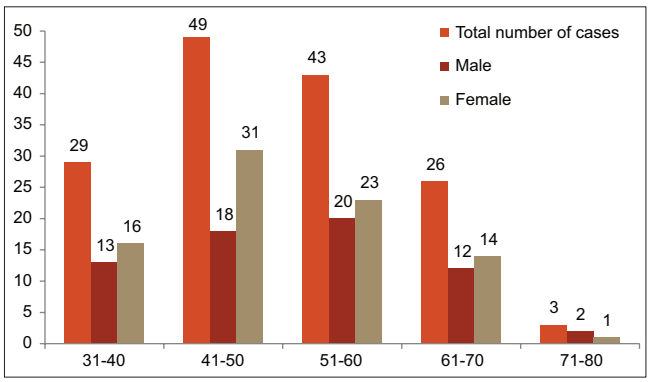

Fig. 1: Age- and gender-wise distribution of cases

Table 1: Pearson's correlation of blood glucose parameters and thyroid function parameters in T2DM obese and non-obese individuals

Pearson's correlation	Correlation	Significant 'p' value
FBS (mg/dl)		
Obese and non-obese	−0.086	0.462
PPBS (mg/dl)		
Obese and non-obese	0.014	0.907
RBS (mg/dl)		
Obese and non-obese	0.220	0.058
HbA1C (%)		
Obese and non-obese	0.216	0.065
T3		
Obese and non-obese	−0.089	0.445
T4		
Obese and non-obese	−0.099	0.398
TSH		
Obese and non-obese	0.065	0.001
BMI		
Obese and non-obese	0.212	0.067

T2DM: Type 2 diabetes mellitus, FBS: Fasting blood sugar, PPBS: Postprandial blood sugar, RBS: Random blood sugar, HbA1C: Hemoglobin A1C, TSH: Thyroid stimulating hormone, BMI: Body mass index

Table 2: Paired "t" test analysis of blood glucose parameters and thyroid function parameters in T2DM obese and non-obese individuals

Paired samples test							
Variables	**Paired differences**					**t**	**Sig. (2-tailed)**
	Mean	**Standard deviation**	**Standard error mean**	**95% confidence interval of the difference**			
				Lower	**Upper**		
FBS (mg/dl)							
Obese and non-obese	2.73333	65.35048	7.54602	−12.30245	17.76911	0.362	0.718
PPBS (mg/dl)							
Obese and non-obese	−23.29333	92.25666	10.65288	−44.51966	−2.06700	−2.187	0.032
RBS (mg/dl)							
Obese and non-obese	−19.34667	42.79160	4.94115	−29.19212	−9.50121	−3.915	0.000
HbA1C (%)							
Obese and non-obese	−2.54865	2.23302	0.25958	−3.06600	−2.03130	−9.818	0.000
T3							
Obese and non-obese	−0.50667	90.82242	10.48727	−21.40301	20.38968	−0.048	0.962
T4							
Obese and non-obese	−0.25467	4.52519	0.52252	−1.29582	0.78649	−0.487	0.627
TSH							
Obese and non-obese	5.2222	0.533	0.21885	−1.74844	1.73511	−0.008	0.000
BMI							
Obese and non-obese	−3.69467	4.39213	0.50716	−4.70520	−2.68413	−7.285	0.000

T2DM: Type 2 diabetes mellitus, FBS: Fasting blood sugar, PPBS: Postprandial blood sugar, RBS: Random blood sugar, HbA1C: Hemoglobin A1C, TSH: Thyroid stimulating hormone, BMI: Body mass index

Table 3: Comparison of blood glucose parameters and thyroid function parameters in T2DM obese and non-obese individuals

Variables	**Mean**	**Standard deviation**	**Standard error mean**
FBS (mg/dl)			
Non-obese	137.1600	39.61403	4.57423
Obese	134.4267	48.67048	5.61998
PPBS (mg/dl)			
Non-obese	159.8267	43.88048	5.06688
Obese	183.1200	81.75903	9.44072
RBS (mg/dl)			
Non-obese	96.6533	22.50435	2.59858
Obese	116.0000	41.67798	4.81256
HbA1C (%)			
Non-obese	5.2932	0.42081	0.04892
Obese	7.8419	2.28575	0.26571
T3			
Non-obese	127.3867	35.18557	4.06288
Obese	127.8933	80.64211	9.31175
T4			
Non-obese	8.4093	2.84259	0.32823
Obese	8.6640	3.25060	0.37535
TSH			
Non-obese	3.5696	4.82576	0.55723
Obese	8.7912	5.3501	0.33838
BMI			
Non-obese	20.7387	2.23582	0.25817
Obese	24.4333	4.28528	0.49482

T2DM: Type 2 diabetes mellitus, FBS: Fasting blood sugar, PPBS: Postprandial blood sugar, RBS: Random blood sugar, HbA1C: Hemoglobin A1C, TSH: Thyroid stimulating hormone, BMI: Body mass index

linked to hypothyroidism. Clinical evidence points to subclinical hypothyroidism, a moderate TD, as a risk factor for overweight and obesity and is connected to considerable changes in body weight [10]. However, this remains a grey area. Little is known about the effect of coexistent Type 2 diabetes mellitus (DM) on thyroid functions in obesity. In the present study, we evaluated the thyroid function and blood glucose parameters in obese Type 2 diabetic patients compared to non-obese Type 2 diabetes individuals. In the present study, the mean values of thyroid hormones T3 and T4 between the obese and non-obese individuals showed that no much significant variations might be due to low sample size. However, the mean values of TSH values were significantly higher 8.7 ng/dl for obese individuals than non-obese individuals. There was a significant positive correlation that was identified for thyroid parameters TSH between obese and non-obese T2DM patients. On the other hand, there was a significant influence of PPBS, RBS, HbA1C, and BMI in obese and non-obese T2DM patients and insignificant influence for thyroid parameters T3 and T4. However, the results also showed that there was a highly significant influence of THS levels in obese T2DM patients. The association between the thyroid functions in obese T2DM patients has only been briefly studied. According to Kouidhi *et al.* (2013) [11], patients who were overweight or obese had considerably higher levels of TSH and FT4 in their blood. These data, however, do not show that these hormones change in diabetes. Mohammed Hussein and AbdElmageed [12] reported similar results. T2DM and thyroid-associated disorders are tightly related. Thyroid problems can make Type 2 diabetes worse, and TD can make diabetes worse. Both T2DM and TD have been demonstrated to be strongly influenced by insulin resistance. Thus, failing to diagnose insulin resistance and low thyroid hormone levels in diabetes can result in subpar patient treatment.

CONCLUSION

The findings of the present investigation showed a high correlation between thyroid function, mainly TSH levels, and Type 2 diabetes, particularly in obese persons. Thyroid problems can make Type 2 diabetes worse, and TD can make diabetes worse. Both T2DM and TD have been demonstrated to be strongly influenced by insulin resistance. Thus, failing to diagnose insulin resistance and low thyroid hormone levels in diabetes can result in subpar patient treatment.

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AUTHORS' CONTRIBUTIONS

All the authors have contributed equally.

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CONFLICTS OF INTERESTS

Declared none.

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