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The Prevalence of Primary Hypothyroidism in Obese Patients in Sana'a City in Yemen

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Abstract

Background and Objective: In many developing countries, including ours, there is often no comprehensive registry for major health problems that contribute to increased mortality and morbidity. Obesity is one such major health problem. The objective of this study was to determine the prevalence of hypothyroidism and associated risk factors in obese patients attending the medical consultation clinic at Al-Kuwait University Hospital (KUH) in Sana'a City, Yemen.

Methods:

This case-control study included a study population of 400 participants. Group A comprised 200 obese individuals, defined according to WHO criteria for obesity, while Group B consisted of 200 non-obese individuals who served as the control group. Both groups underwent a comprehensive history and physical examination, which included measurements of BMI and blood pressure. Laboratory analyses performed included fasting blood sugar (FBS), lipid profile, free T3, free T4, and thyroid-stimulating hormone (TSH).

Results: During the study period, 400 participants were included and divided into two groups: Group A (case group) consisting of 200 obese patients, and Group B (control group) consisting of 200 non-obese individuals. The prevalence of hypothyroidism in Group A was 20%, with 26% of these patients being male and 74% female. In Group B, the prevalence of hypothyroidism was 10%. Obese patients exhibited a higher prevalence of hypertension, elevated fasting blood sugar (FBS), and abnormal lipid profiles compared to the non-obese group. Additionally, obese hypothyroid patients had a higher prevalence of hypertension, diabetes mellitus (DM), and abnormal lipid profiles compared to obese euthyroid patients.

Conclusion: The prevalence of hypothyroidism in our study is high among obese and overweight patients, similar to findings in many other countries. It is strongly associated with type 2 diabetes, hypertension, elevated triglycerides, high LDL cholesterol, and low HDL cholesterol.

Introduction

Obesity and overweight are significant risk factors for disability-adjusted life years (DALYs) and mortality. In 2015, obesity was associated with approximately 4 million deaths and 120 million DALYs, accounting for 7.1% and 4.9% of all global deaths and DALYs, respectively. Over the past thirty years, the prevalence of obesity has surged globally. As of the most recent estimates, the global prevalence of obesity among adults is approximately 12%, with a notably higher prevalence observed in women compared to men(1,2)

Thyroid disease represents a significant global health concern with substantial implications for overall health and well-being. Hypothyroidism, a prevalent thyroid disorder, occurs more frequently in women than in men. This disparity underscores the importance of addressing thyroid dysfunction, particularly in the female population, to mitigate its impact on health(3)

Body Mass Index (BMI) is a statistical measure used to estimate body fat based on an individual's weight and height, applicable to both males and females across all ages. It is calculated by dividing a person's weight in kilograms by the square of their height in meters, expressed as BMI = weight (kg) / height^2 (m^2). The resulting value is the individual's BMI. The National Institutes of Health (NIH) utilizes BMI to classify individuals into categories such as underweight, normal weight, overweight, or obese, replacing traditional height versus weight charts. These BMI classifications are employed by both the NIH and the World Health Organization (WHO) for diverse populations, including White, Hispanic, and Black individuals(4)

The Body Mass Index (BMI) categories are defined as follows:

Severely Underweight**: BMI less than 16.5 kg/m^2

Underweight**: BMI less than 18.5 kg/m^2

Normal Weight**: BMI ranging from 18.5 to 24.9 kg/m^2

Overweight**: BMI ranging from 25 to 29.9 kg/m^2

Obesity**: BMI greater than or equal to 30 kg/m^2

Obesity Class I**: BMI ranging from 30 to 34.9 kg/m^2

Obesity Class II**: BMI ranging from 35 to 39.9 kg/m^2

Obesity Class III: BMI greater than or equal to 40 kg/m^2 (also

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referred to as severe, extreme, or massive obesity)(4)

Hypothyroidism is characterized by clinical manifestations such as lethargy, weight gain, constipation, and other symptoms. It is diagnosed based on laboratory findings showing elevated Thyroid-Stimulating Hormone (TSH) levels (>4.2 μIU/ml) coupled with reduced levels of Free T3 and Free T4.

In contrast, subclinical hypothyroidism (SCH) is defined biochemically by elevated serum TSH levels (4.2–9.9 μIU/ml) while Free T3 and Free T4 levels remain within the normal range. Notably, SCH often presents asymptomatically, and the diagnosis is generally made through routine thyroid function tests in patients who do not exhibit overt clinical symptoms.

SCH has been identified as a potential risk factor for cardiovascular diseases, particularly atherosclerosis and myocardial infarction, in elderly women. The prevalence of SCH increases with age, especially among females, underscoring the importance of regular thyroid function screening in older populations. Although SCH does not typically necessitate immediate intervention, ongoing monitoring and evaluation of thyroid function and cardiovascular risk factors are advisable.

A significant proportion of patients with subclinical hypothyroidism progress to overt hypothyroidism annually, with an incidence rate ranging from 4.3% to 8%. This progression is notably more common among the elderly, who exhibit a higher predisposition to developing overt hypothyroidism(5)

Thyroid dysfunctions, following diabetes, rank as the second most prevalent metabolic disorders globally. The prevalence of thyroid dysfunction varies significantly across different regions, influenced by a range of factors including biological, environmental, and geographical elements.

Obesity is associated with various physiological and pathological changes affecting multiple body organs, including the thyroid gland. Although numerous studies have explored the relationship between obesity and thyroid dysfunction, the findings remain inconsistent. Most research indicates a positive association between Body Mass Index (BMI) and serum Thyrotropin (TSH) levels, as well as an increased prevalence of hypothyroidism among obese individuals. (7-11) Conversely, the relationship between BMI and serum-free thyroid hormone levels is more contentious. Some studies have reported a negative association between BMI and Free Thyroxin (FT4), while others have observed either a positive association or no significant relationship between BMI and FT4 levels (8,9,12-15)

Hypothyroidism is linked to reduced thermogenesis and a lower metabolic rate, which contribute to its association with increased Body Mass Index (BMI) and a higher prevalence of obesity. These metabolic alterations underscore the connection between hypothyroidism and weight gain(16).

Adipose tissue, or fat cells, function as an active endocrine organ by producing leptin (17,18) The correlation between Thyrotropin (TSH) and Body Mass Index (BMI) may be mediated by leptin, which is secreted by fat cells. Leptin plays a crucial role in regulating energy homeostasis by signaling the central nervous system about the body's adipose reserves (17). It modulates neuroendocrine and behavioral responses to overfeeding, thereby influencing both food intake and energy expenditure.

Leptin functions as a crucial neuroendocrine regulator of the hypothalamic-pituitary-thyroid (HPT) axis. It influences the regulation of Thyrotropin-Releasing Hormone (TRH) gene expression in the paraventricular nucleus of the hypothalamus(18,19). In turn, Thyrotropin (TSH) stimulates the secretion of leptin from human adipose tissue (19,20,21). Additionally, leptin modulates thyroid deiodinase activities, which affects the conversion of thyroxine (T4) to triiodothyronine (T3)(17,22). These interactions support the concept of an inverse relationship between thyroid hormones and leptin.

There is a paucity of data from Yemen regarding non-communicable diseases such as obesity, diabetes mellitus (DM), cardiovascular diseases (CVD), and hypothyroidism(23,24,25), Specifically, there are no published population-based studies investigating the prevalence of hypothyroidism among individuals with obesity. To address this gap, we designed this study to determine the prevalence of hypothyroidism and its associated risk factors among Yemeni patients with obesity. This research aims to stimulate further investigations into the prevalence of hypothyroidism in both the general population and among individuals with obesity in Yemen.

Materials and Methods

This case-control study utilized a sampling frame that included both nationals and non-nationals, aged 17 to 70 years, of both genders, attending Al-Kuwait University Hospital (KUH) and the consultation clinic in Sana'a City. A total of 400 participants were enrolled in the study, divided into two distinct groups:

- Group A: The patient group, consisting of individuals classified as overweight or obese according to the WHO criteria for obesity.
- Group B: The control group, comprising individuals with normal weight as defined by the same WHO criteria.

The study aimed to compare the prevalence of hypothyroidism and associated risk factors between these two groups.

The study received approval from the Joint Ethics Committee of the Faculty of Medicine and Health Sciences at Sana'a University. It was conducted from May 2020 to May 2022. Participants who were pregnant, had secondary hypothyroidism, thyroid carcinoma, or significant euthyroid diseases were excluded from the study.All participants underwent a comprehensive evaluation that included a detailed medical history and physical examination. Measurements recorded included height (taken without shoes), weight (measured while wearing indoor clothing), the presence of goiter, peripheral signs of dyslipidemia, and blood pressure. Body Mass Index (BMI) was calculated using the formula: weight (kg) divided by height (m^2). Participants were classified into categories based on their BMI as follows:

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- Normal weight**: BMI ≤ 24.9 kg/m^2
- Overweight**: BMI ranging from 25 to 29.9 kg/m^2
- Obesity**: BMI ranging from 30 to 39.9 kg/m^2
- Morbid obesity**: BMI ≥ 40 kg/m^2 (4)

Blood pressure (BP) was measured using a mercury sphygmomanometer following a 10-minute rest period, with the patient seated. Each participant's BP was recorded twice, with measurements taken at a 5-minute interval. The first and fifth Korotkoff sounds were used to determine systolic and diastolic blood pressure, respectively. The second measurement was used as the final recorded blood pressure for each individual.

Hypertension was defined according to the 2018 ESC/ESH guidelines for the management of arterial hypertension: systolic blood pressure of 140 mmHg or higher and/or diastolic blood pressure of 90 mmHg or higher (26), or if the patient was undergoing treatment with antihypertensive medications.

Laboratory parameters, including fasting blood sugar (FBS), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), and thyroid function tests, were measured directly using the Roche/Hitachi 911 Automated Chemistry Analyzer. Diabetes mellitus (DM) was diagnosed based on World Health Organization (WHO) criteria (27) or if the participant was currently using anti-diabetic medications.

Dyslipidemia was assessed based on the updated National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) guidelines [28]. The study included both preexisting and newly detected cases of primary hypothyroidism, which were further classified into:

- Subclinical Hypothyroidism**: Characterized by elevated TSH levels with normal Free T3 (FT3) and Free T4 (FT4) levels.
- Overt Hypothyroidism**: Defined by elevated TSH levels with low FT3 and FT4 levels.

Thyroid function tests were conducted using an enzyme-linked immunosorbent assay (ELISA). The reference values in our laboratory were: TSH 0.4-4.4 µIU/ml, FT4 0.8-1.8 ng/dl, and FT3 2-4.4 µIU/ml. Thyroid dysfunction was diagnosed according to the guidelines set forth by the American Thyroid Association [29]. Statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS) version 28.0. Differences between groups were assessed using the chi-square test for categorical variables and the t-test for numerical variables. A p-value of ≤ 0.05 was considered statistically significant.

Results

4oo participants were included in this study which was classified into two groups (group A or the patient group =200 patients they are overweight or obese, 52 (26%) were males and 148 (74%) were females and there age ranges between 17-70 years with mean 54.3±34)

and (group B or the control group with normal weight, 61 (30.5%) were males and 139(69.5%) were females and their age ranges between 18-70 with mean 53.21±32 SD)

The prevalence of hypothyroidism in the obese group was 40 cases (20%), with 10 males (25%) and 30 females (75%). Among these cases, 25 (12.5%) were diagnosed with overt hypothyroidism and 15 (7.5%) with subclinical hypothyroidism. In the control group, the prevalence of hypothyroidism was 20 cases (10%), including 13 cases (6.5%) of overt hypothyroidism and 7 cases (3.5%) of subclinical hypothyroidism.The difference in the prevalence of hypothyroidism between the obese and control groups was highly significant, with a p-value ≤ 0.005. There were significant differences between Group A and Group B in several clinical parameters:

High Systolic and Diastolic Blood Pressure**: 37.5% in Group A vs. 15% in Group B,Type 2 Diabetes Mellitus 26% in Group A vs. 5% in Group B

High Serum Triglycerides (TG)**: 19% in Group A vs. 7% in Group B

- Low Serum High-Density Lipoprotein (HDL)**: 18.5% in Group A vs. 7.5% in Group B
- High Serum Low-Density Lipoprotein (LDL)**: 16.5% in Group A vs. 5.5% in Group B.These differences were statistically significant, with a p-value ≤ 0.005

Table 1: Clinical and Laboratory Characteristics of Obese and Control Groups

Factors	Total 400	With obesity=200	Without obesity=200	p-value
Age	52.11±21	54.3±34	53.21±34	0.05
Male sex%	113(28.2%)	52(26%)	61	0.34
Type 2- DM %	62 (15.5%)	52(26%)	10(5%)	≤0.005
High BPmmHG	105 (26.2%)	75(37.5%)	30(15%)	≤0.005
High TGmg|dl	52(13%)	38(19%)	14(7%)	0.005
Low HDLmg|dl	52(13%)	37(18.5%)	15(7.5%)	0.005

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High LDLmg|dl	44(11%)	33(16.5%)	11(5.5%)	0.0006
Hypothyroidism				
Total	65(16.2%)	40(20%)	20 (10%)	0.007
Frank	40(10%)	25(12.5%)	13 (6.5%)	0.05
Subclinical	25(6.2%)	15(7.5%)	7(3.5%)	0.12

Table 2: The clinical and laboratory characteristics of obese patients with and without hypothyroidism.

Factors	Total=200	With hypothyroidsm =65	Without hypothyroidism=135	P=value
Age in years	53.2±21	54.3±32	52.6±41	0.5
Male sex%	53(26.5%)	10(15.3%)	43(31.8%)	0.002
Type 2- DM %	52(26%)	20 (30%)	30(22.2%)	0.001
High BPmm|HG	75(37.5%)	27(41.5%)	48(35.5%)	0.003
Serum TG mg|dl	52(26%)	23 (35.3%)	29(21.4%)	0.001
Serum HDLmg|dl	52(26%)	25(38.3%)	27 (20%)	0.005
Serum LDLmg|dl	44(22%)	20 (30.7%)	24 (17.7%)	0.001

Discussion

In this study, the aim was to analyze this prevalence prevalence of primary hypothyroidism in over weight and obese patients who attended the medical department and consultation clinic in KUH using the WHO definition of obesity [4] and the American Thyroid Association (ATA) criteria for diagnosing thyroid dysfunction[29]

Previous research has consistently shown a higher prevalence of primary hypothyroidism among obese individuals, with reported rates ranging from 13.2% to 44% [18, 19, 20, 21]. A notable contrast is seen in the study from the United Arab Emirates (UAE), where the prevalence of primary hypothyroidism among obese patients was reported to be only 8.8% [30]. This lower prevalence might be influenced by various factors, such as differences in population demographics, dietary habits, healthcare practices, or environmental factors

To my knowledge this is the first study about the prevalence of hypothyroidism among obese patients in Yemen and its results provide invaluable information on the hypothyroidism in such high-risk group and should encourage researchers to investigate more such an emerging, health problem. The overall prevalence of hypothyroidism among over weight and obese patients in the present study found to be 20% of which 12.5% had frank and 7.5% had subclinical hypothyroidism which is similar to the above-mentioned studies [18,19,20,21

This study demonstrates that the prevalence of hypothyroidism is significantly higher in obese individuals (20%) compared to non-obese individuals (10%). This finding is consistent with the study by Nagila et al., which reported that thyroid-stimulating hormone (TSH) levels were significantly elevated in obese patients relative to non-obese controls (22). However, this result contrasts with the study conducted in the United Arab Emirates (UAE), where elevated TSH levels were observed in only 8.8% of the obese population (30).

The association between obesity and hypothyroidism has been documented in various studies (11, 31). This relationship may be attributed to the elevation of TSH levels observed in obese patients (31). The elevated TSH levels could reflect an underlying hypothyroid condition that is more prevalent among the obese, potentially due to metabolic or inflammatory factors associated with excess body weight.

TSH levels are often at the upper limit of the normal range or slightly elevated in obese children, adolescents, and adults. This increase in TSH is positively correlated with body mass index (BMI) (11, 31, 32, 33, 34). Evidence suggests that TSH levels are positively associated with the degree of obesity, indicating that higher levels of TSH are observed as the degree of obesity increases (11). This correlation underscores the potential impact of obesity on thyroid function and highlights the importance of monitoring thyroid hormone levels in obese individuals.

A positive correlation has been observed between serum leptin levels and serum TSH levels in obese individuals (11). This relationship may reflect the positive association between TSH levels and body mass index (BMI) reported in some studies (17).

The mechanism underlying elevated TSH levels in obesity can be elucidated by the increased leptin hormone levels observed in obese patients. Leptin, which is secreted by peripheral adipose tissue, acts directly on the paraventricular nucleus of the hypothalamus. This action stimulates the release of thyrotropin-releasing hormone (TRH) from the hypothalamus, leading to an increase in TSH secretion by the pituitary gland. Additionally, neurotransmitters and hormones that regulate body weight and satiety may also play an indirect role in modulating TSH production. This complex interaction underscores the multifaceted nature of hormonal regulation in obesity and its impact on thyroid function.

Increased levels of leptin and pro-opiomelanocortin (POMC) contribute to the downregulation of agouti-related peptide

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(AgRP). This regulatory change leads to the activation of melanocyte-stimulating hormone (MSH) and subsequent stimulation of thyrotropin-releasing hormone (TRH) neurons, resulting in elevated TSH levels. Additionally, elevated TSH levels have been associated with severe obesity. This association may be attributed to the direct action of TSH, which stimulates preadipocyte differentiation and promotes adipogenesis, thereby exacerbating the obesity condition (35). This interplay highlights the complex mechanisms linking thyroid function and adiposity.

In our study, the prevalence of individual metabolic comorbidities among obese hypothyroid patients was notably high. Hypertension was the most frequently observed condition, followed by dyslipidemia—characterized by elevated serum triglycerides (TG), decreased high-density lipoprotein (HDL) cholesterol, and increased low-density lipoprotein (LDL) cholesterol—and diabetes mellitus (DM). These findings are consistent with those reported in previous studies (36, 37, 38, 39, 40). Specifically, the prevalence of hypertension among hypothyroid patients in our study was 41.5%, reflecting a significant association between hypothyroidism and increased risk of hypertension.

Hypothyroidism is well-recognized as a potential cause of secondary hypertension. Previous studies have consistently demonstrated elevated blood pressure values in individuals with hypothyroidism (36, 37, 40). The pathophysiological link between hypothyroidism and diastolic hypertension may involve increased peripheral vascular resistance and reduced cardiac output. Additionally, hypothyroid patients often exhibit significant changes in blood volume, which may trigger a volume-dependent mechanism of blood pressure elevation characterized by low plasma renin activity (41, 42). These findings highlight the complex interplay between thyroid dysfunction and hypertension, emphasizing the need for careful management of blood pressure in hypothyroid patients

The prevalence of diabetes mellitus (DM) was notably higher in obese hypothyroid patients (30%) compared to obese patients without hypothyroidism (22.2%). This increased prevalence underscores the significant metabolic impact of hypothyroidism in the context of obesity, suggesting a potential exacerbation of glycemic dysregulation in hypothyroid individuals.

The association between thyroid disease and glucose metabolism is well-documented (43). Insulin resistance and hyperinsulinemia are critical components of metabolic syndrome and are commonly observed in conjunction with obesity (44). This relationship underscores the complex interplay between thyroid dysfunction, insulin sensitivity, and overall metabolic health.Insulin resistance has been related with both ends of the thyroid dysfunction spectrum, although the poorly understood mechanisms might be different in each case (45) The explanation for this relationship could be an unopposed activation of gluconeogenesis (45). Moreover, hyperthyroidism, even in subclinical forms, is associated with a reduced insulin half-life, most likely because of accelerated insulin degradation (43,44,45). On the other hand, hypothyroidism-associated insulin resistance could be the result of diminished tissue sensitivity to insulin. Accordingly, glucose disposal is then reduced in this situation (43). In hypothyroidism, insulin resistance is counterbalanced by a parallel reduction in gluconeogenesis, making it habitually irrelevant without clinical consequences (43).

Hypothyroidism, like obesity, is one of the pathological conditions most frequently associated with disorders of lipid metabolism, which can lead to dyslipidemia—a major risk factor for coronary disease. Overt hypothyroidism is characterized by hypercholesterolemia and a marked increase in LDL cholesterol due to decreased fractional clearance of LDL, which is caused by a reduced number of LDL receptors in the liver.

However, the controversy persists regarding the lipids level in subclinical hypothyroidism and its clinical significance. Moreover, it is likely to be a risk factor for atherosclerosis and coronary diseases.(46,47,48,49). In this study, the prevalence of dyslipidemia was found to be higher in obese hypothyroid patients.

Conclusion

The prevalence of hypothyroidism in our study is high among obese and overweight patients, consistent with findings from many other countries. Hypothyroidism is strongly associated with type 2 diabetes, hypertension, elevated triglycerides, and LDL cholesterol, along with low HDL cholesterol. We recommend evaluating thyroid function in any obese or overweight patient, as managing hypothyroidism can help control several risk factors for cardiovascular disease.

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