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ORIGINAL ARTICLE

Prevalence of subclinical hypothyroidism in local adult obese population.

Afrah Malik[1], Muhammad Tahir[2], Muhammad Zuhair Tahir[3], Muhammad Zubair Tahir[4], Ayesha Salim[5], Munir Ahmad[6]

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ABSTRACT… Objective: To find out the frequency of subclinical hypothyroidism (SCH) in local adult obese population and to determine the risk of cardiovascular events in patients with SCH using the 10-year Atherosclerotic Cardiovascular Disease (ASCVD) risk calculator. **Study Design:** Cross Sectional & Descriptive study design. **Setting:** Medical OPD, PAF Hospital, Islamabad. **Period:** 20th July 2023 to 20th January 2024. **Methods:** A total of 200 patients of both gender with Body Mass Index (BMI) greater than 28kg/m^2 were included in the study. Blood samples were Collected from all patients and sent to the hospital laboratory for performing thyroid function tests and fasting lipid profile. Patients found to have SCH (TSH levels more than reference range with normal T3 and T4 levels) had their ten-year risk of cardiovascular events determined using the ASCVD Risk Calculator. A calculated 10 year ASCVD risk of ≥7.5% was concluded significant. **Results:** Age range in this study was from 20 to 65 years with mean age of 43.295±8.85 years, mean BMI 30.960±1.53 Kg/m^2, mean TSH 2.904±1.29 mIU/L (normal range 0.27 to 4.2 mIU/ml), mean T3 103.230±6.86 ng/dL (normal range 94 to 170 ng/dL) and mean T4 was 10.092±1.26 μg/dL (normal range 5 to 11 μg/dL). Subclinical hypothyroidism was observed in 16% patients. Amongst these patients, increased cardiovascular event risk was present in 25% patients. **Conclusion:** Our study has concluded a high prevalence of subclinical hypothyroidism among obese adult patients and increased risk of cardiovascular events in patients with SCH.

Key words: Cardiovascular Events, Obesity, Subclinical Hypothyroidism.

INTRODUCTION

The thyroid gland regulates a metabolic equilibrium among all vital organs of the human body mediated by the pituitary and hypothalamus. The thyroid is "stimulated" by Thyroid Stimulating Hormone (TSH) to make and release its two primary hormones, T3 and T4 (free thyroxine). Levels of T3 and T4 are inversely related to the release of thyroid stimulating hormone from pituitary gland. TSH released by the pituitary and T3 and T4 concentrations in the blood have an inverse relationship with each other. Under normal conditions of health there is a critical balance between the two, with a finely tuned feedback mechanism. When there is not enough T3 or T4 circulating in the body, more TSH is released; when there is enough or too much thyroid hormone circulating in the peripheral blood, the pituitary releases less TSH.[1] Subclinical hypothyroidism (SCH) is characterized by a serum thyroid-stimulating hormone (TSH) level that exceeds the upper limit of normal, while serum free thyroxine (T4) levels remain within the normal range.[2]

Although the link between obesity and subclinical thyroid disorders is yet unknown, the linkage between obesity and hypothyroidism is widely recognized.[3] Globally, obesity is become a serious public health issue. Asians have a higher BMI than Europeans, according to studies. As a result, the international obesity task force recommends that Asians have a BMI of 23.0-24.9 kg/m2 for overweight people and >25.0 kg/m2 for obese people.[4]

Both obesity and SCH have quite a few common adverse outcomes. These include dyslipidemia, high blood pressure, insulin resistance, and coronary heart disease.[5] Thyroid auto antibodies, also known as thyroglobulin antibodies, have been

1. MBBS, Post Graduate Resident Medicine, PAF Hospital, Islamabad.
2. MBBS, FCPS, FACP, FRCP (Glasg), Professor & Head Medicine, PAF Hospital, Islamabad.
3. MBBS, Medical Officer Medicine, PAF Hospital, Islamabad.
4. MBBS, Post Graduate Medicine, PAF Hospital, Islamabad.
5. MBBS, Post Graduate Resident Medicine, PAF Hospital, Islamabad.
6. MBBS, FCPS (Medicine), FCPS (Cardiology), Associate Professor Cardiology, Faisalabad Institute of Cardiology, Faisalabad.

Correspondence Address:
Dr. Afrah Malik
Department of Medicine
PAF Hospital, Islamabad.
afra_028@hotmail.com

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linked to obesity and SCH, according to a Chinese study, in which Obesity was explained as a body mass index (BMI) of 28 kg/m^2 or more.[5] Another study attempted to establish the relationship between obesity, metabolic syndrome, SCH and thyroid autoimmunity (TAI). The study concluded that obesity is a risk factor for SCH and TAI. Additionally, the impact of obesity on thyroid diseases was found to be more pronounced in patients with metabolic syndrome.[3] High-sensitive TSH (hsTSH) levels within the upper limits of the normal range have been linked to an high risk of central obesity, insulin resistance, inflammation, hyperlipidemia, hyperuricemia, hypercoagulability, hyperglycemia, and metabolic syndrome. These findings suggest that hs TSH is a new biomarker for cardiometabolic risk.[2] A comparable study conducted in Peshawar revealed that 15% of the adult obese population there had SCH.[4] A cross-sectional adult survey conducted in India regarding the effects of SCH on the cardiovascular system revealed no correlation between SCH and the Framingham 10-year risk of cardiac events.[6] A cross-sectional comparative study was conducted in Lahore between male myocardial infarction (MI) patients and age-matched controls to evaluate the existence of SCH and other clinical risk factors. MI patients had an incidence of SCH of 12%, while healthy control participants had an incidence of 5%. No statistically significant difference was observed between the two groups ($p=0.15$). The investigation revealed no connection between MI and SCH.[7]

This study aimed to explore the association between BMI and subclinical hypothyroidism, given the paucity of data on the condition in the adult obese population in Islamabad. Using the 10-year Atherosclerotic Cardiovascular Disease (ASCVD) Risk Calculator, the cardiovascular risk of those individuals who were diagnosed with SCH was determined. This may help patients by detecting and managing risk factors, improving prognosis, and avoiding complications.

METHODS

This study was carried out during a six-month period, from July 20, 2023, to January 20, 2024, at the Department of Medicine, PAF Hospital, Islamabad. It is cross-sectional and descriptive in nature. The study was approved by the hospital's ethical review committee (MED-2021-258-17896). Using the WHO sample size calculator, the sample size was determined to be 200 with a 95% confidence level.[4] The sampling method used was non-random, consecutive collection. The study included all patients aged 20–65 with a BMI more than 28 kg/m2 who visited the medical OPD for a routine checkup throughout the specified timeframe. Patients with preexisting thyroid abnormalities, cardiovascular conditions (stroke or ischemic heart disease), renal or hepatic failure, thyroid medication users, and women who were pregnant or nursing were excluded from the study. The patients provided written informed consent. The factors, which included age, gender, BMI, comorbidities (diabetes, hypertension, dyslipidemia, cardiovascular disease), history of any autoimmune illnesses, family history of thyroid problems, history of smoking, and physical activity, were evaluated using a self-designed questionnaire. Every patient had blood drawn, which was then forwarded to the hospital laboratory for fasting lipid profile and thyroid function tests (TFTs). Patients' blood pressure was also taken.

The ASCVD Risk Calculator, which considers factors such as age, sex, race, blood pressure, total and HDL cholesterol levels (mg/dl), history of diabetes, smoking, and antihypertensive medication, was used to calculate the ten-year risk of cardiovascular events for the individuals who were found to have SCH. A 10-year ASCVD risk estimate of $\geq 7.5\%$ was considered significant. With SPSS version 23, data analysis was carried out. For quantitative data, such as age, BMI, and thyroid function test results (serum TSH, T3, and T4 levels), mean and standard deviation (SD) were derived. For categorical qualities such gender, employment position, physical activity, smoking history, comorbidities, family history, and subclinical hypothyroidism; frequencies and percentages were calculated. Using the post-stratified Chi square test, effect modifiers including age, gender, comorbidities, family history, and smoking history were stratified. A p-value of 0.05

or less was regarded as significant.

RESULTS

All the participants of this study had age between 20 to 65 years. The average age was 43.295±8.85 years, with a mean BMI of 30.960±1.53 Kg/m2, a mean TSH of 2.904±1.29 mIU/L, a mean T3 of 103.230±6.86 ng/dL, and a mean T4 of 10.092±1.26 µg/dL. Of the patients, 28.5% were female and 71.5% were male. Of 200 individuals, 16% had subclinical hypothyroidism (32 out of 200 cases). 25% of the patient population (8 out of 32 patients) had a cardiovascular event risk due to subclinical hypothyroidism. The 10-year calculated risk of ASCVD for each of these 25% of patients is displayed in Figure-1.

Table-I displays the stratification of cardiovascular event risk and SCH among patients with SCH based on age, gender, comorbidities, smoking history, and family history.

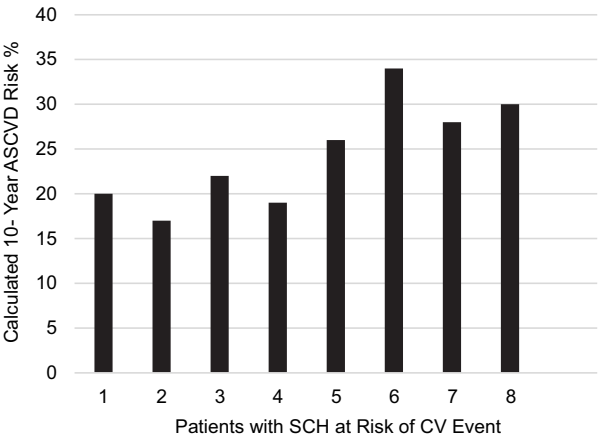

Figure-1

Factors	SCH		P-Value	Cardiovascular Event Risk in SCH		P-Value
	Yes	No		Yes	No	
Age (years)						
20-40	8(10.5%)	68(89.5%)	0.098	4(50%)	4(50%)	0.059
41-65	24(19.4%)	100(80.6%)		4(16.7%)	20(83.3%)	
Total	32(16%)	168(84%)		8(25%)	24(75%)	
Gender						
Male	23(16.1%)	120(83.9%)	0.959	6(26.1%)	17(73.9%)	0.820
Female	9(15.8%)	48(84.2%)		2(22.2%)	7(77.8%)	
Total	32(16%)	168(84%)		8(25%)	24(75%)	
Comorbidities						
None	12(11.1%)	96(88.9%)	0.115	2(16.7%)	10(83.3%)	0.202
Diabetes	9(33.3%)	18(66.7%)		5(55.6%)	4(44.4%)	
Hypertension	5(16.1%)	26(83.9%)		0(0%)	5(100%)	
Dyslipidemia	4(18.2%)	18(81.8%)		1(25%)	3(75%)	
Cardiovascular disease	1(11.1%)	8(88.9%)		0(0%)	1(100%)	
Autoimmune Diseases	1(33.3%)	2(66.7%)		0(0%)	1(100%)	
Family History						
Yes	12(66.7%)	6(33.3%)	0.000	1(8.3%)	11(91.7%)	0.092
No	20(11%)	162(89%)		7(35%)	13(65%)	
Smoking						
Yes	10(21.7%)	36(78.3%)	0.226	3(30%)	7(70%)	0.660
No	22(14.3%)	132(85.7%)		5(22.7%)	17(77.3%)	

Table-I Stratification of results: The Chi Square Analysis

DISCUSSION

In our study, the mean BMI was 30.960±1.53 kg/m^2 with mean TSH 2.904±1.29 mIU/L, mean T3 103.230±6.86 ng/dl and mean T4 was 10.092±1.26 µg/dL. Subclinical hypothyroidism was seen in 16% of patients. Similar to these results, meta -analysis by Michalaki et al. also reported a high rate of dysfunction of thyroid in obese patients with increased TSH levels.[8] The metabolism of glucose is significantly influenced by thyroid hormones. Obesity, hyperglycemia, insulin resistance, dyslipidemia, and an elevated risk of cardiovascular events are linked to significant hypothyroidism.[9] Given that SCH is more common in obese patients than in non-obese patients, the impact of thyroid hormones on metabolic markers is investigated. Several studies have also looked into the association between thyroid hormones and the metabolism of fat and glucose in obese young adults and children.[9-13]

Lipid production, metabolism, and mobilization are all affected by thyroid hormones. Several studies demonstrated a significant connection between TG or TC and TSH levels.[10] Our study revealed a negative link with HDL-C levels and a positive correlation with TSH and triglyceride levels in the SCH group. In SCH patients, we observed a greater prevalence of hyperlipidemia. Triglyceride levels rise and HDL falls as insulin resistance worsens. One possible explanation is that elevated TG levels facilitate the easier catabolization of HDL particles. Lower plasma HDL-C is a result of both increased neutral lipid transfer and decreased lipoprotein lipase (LPL) activity. [14] This mechanism could account for the elevated TG and low HDL-C values observed in our study of SCH patients.

In this study, 25% of patients with SCH had an increased ASCVD event risk. There is evidence from several studies that SCH and CVD are correlated. An observational study in Netherlands revealed that SCH-afflicted postmenopausal women were more likely to suffer a myocardial infarction (MI) (OR 2.3, 95%, CI 1.3–4.0). Those with positive serum TPO antibodies were at a substantially higher risk (OR 3.5, 95%, CI 1.7–7.4). Aortic atherosclerosis was more common in women with SCH regardless of their status of TPO antibodies. The study's estimated 14% resulting from risk of subclinical hypothyroidism to MI was comparable to the 14%–18% calculated risks for smoking, hypertension, diabetes, and hypercholesterolemia.[15] Biochemical SCH was linked to an elevated risk of CVD in a Danish primary care population of 1,212 patients age between 20 to 69 years lacking a history of thyroid disease, but exclusively in men under 50 years old (OR 3.3, 95%, CI 1.6–6.8).[16] The existence of SCH may raise the risk of cardiovascular consequences in people with higher baseline CVD risks. Compared to patients who were euthyroid, cardiac disease history with SCH who were hospitalized in a hospital of Italy had a 2.4-times high risk of death due to cardiac disease.[17] The Cleveland Clinic Preventive Cardiology Clinic studied individuals at high risk for ASCVD, and the results showed that untreated patients, those under 65, and those having serum thyroid stimulating hormone levels between 6.1–10 mIU/L or more than 10 mIU/L had higher all-cause mortality.[18]

The Thyroid Studies Collaboration carried out a large pooled analysis of individual participants from prospective studies (n = 55,287) and discovered that adults with Thyroid Stimulating Hormone levels between 10–19.9 mIU/L had a higher risk of coronary heart disease and SCH compared to adults with normal TSH levels. The risk was greater with increasing serum TSH levels (P for trend <0.001).[19] The Thyroid Studies Collaboration also performed a large pooled analysis involving approximately 31,900 participants, which showed that the high risk of coronary heart disease cases in SCH patients was unaffected by the presence or absence of serum anti-TPO antibodies (P for interaction = 0.65). Additionally, according to the latest work and comprehensive meta-analysis by Moon et al., individuals with Subclinical Hypothyroidism faced a 33% higher risk of cardiovascular disease compared to those with normal thyroid function.[21]

However, some research has failed to show a conclusive connection between Subclinical Hypothyroidism and CVD. A study on health of

cardiovascular system was a large prospective cohort study included 3,233 older people, of which 496 (15%) had SCH.[22] In terms of coronary heart disease prevalence, patients with Subclinical hypothyroidism (19.8%) and with euthyroid (18.5%) did not differ.[22] A follow-up research by Hyland KA et al., with a sample size of 4,184 euthyroid people and 679 with SCH, found no significant relation between cardiovascular events and SCH.[23]

CONCLUSION

The results of our study show that obese adult patients have a high prevalence of subclinical hypothyroidism, which raises serious concerns about this population. Furthermore, a significant fraction of individuals diagnosed with subclinical hypothyroidism exhibited an elevated 10-year risk of cardiovascular events. When considered collectively, these findings point to a link between obesity, subclinical hypothyroidism, and a higher risk of cardiovascular morbidity. If diagnosing and treating mild thyroid dysfunction can lower cardiovascular risk in obese people, more study has to be done on this topic. However, these results imply that subclinical hypothyroidism may be linked to unfavorable health outcomes and should be identified and treated early in the management of obesity.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION

No.	Author(s) Full Name	Contribution to the paper	Author(s) Signature
1	Afrah Malik	Conception & design of study, acquisition, analysis and interpretation of data, Manuscript writing.	
2	Muhammad Tahir	Conception & design of study, acquisition, analysis and interpretation of data, Manuscript writing, Critical Reivew.	
3	Muhammad Zuhair Tahir	Conception & design of study, acquisition, analysis and interpretation of data, Manuscript writing.	
4	Muhammad Zubair Tahir	Conception & design of study, acquisition, analysis and interpretation of data, Manuscript writing.	
5	Ayesha Salim	Conception & design of study, acquisition, analysis and interpretation of data, Manuscript writing.	
6	Munir Ahmad	Analysis and interpretation of data, Manuscript writing.	