DOI: https://doi.org/10.53350/pjmhs020241811083

ORIGINAL ARTICLE

Association Between Thyroid-Stimulating Hormone Levels and Metabolic Syndrome in Postmenopausal Women

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ABSTRACT

Background: After menopause, metabolic syndrome (MetS) is prevalent, and is linked to elevated cardiovascular and metabolic risk. Even tiny changes in thyroid function can help to cause these abnormalities within the euthyroid range. The relationship between MetS and serum levels of thyroid-stimulating hormone (TSH) was examined in this study.

Material and Methods: This analytical cross-sectional study included 120 postmenopausal women recruited from Sindh Government Hospital Saudabad, Karachi, and the Department of Obstetrics and Gynaecology, Sindh Rangers Hospital, Pakistan, from June 2022 to June 2023. Anthropometric measurements, blood pressure, fasting plasma glucose, lipid profile, TSH & free thyroxine were evaluated. Harmonized criteria were used for diagnosis of MetS. The correlation analysis and multivariable logistic regression model were employed to examine associations.

Results: MetS was present in 59 participants (49.2%). The median TSH level was significantly higher in women with MetS compared to women without MetS (2.61 vs. 1.79 mIU/L; p<0.001). The prevalence of MetS rose with each quartile of TSH levels, ranging from 30.0% in the lowest quartile to 73.3% in the highest quartile, with a p value of less than 0.001 for trend. TSH had positive correlation with waist circumference, BMI, triglycerides, blood pressure, fasting glucose and negative correlation with HDL-C. Multivariate analysis showed that women in the highest quartile of TSH level had higher risk for MetS compared with the lowest quartile of TSH level (adjusted OR 3.15; 95% CI 1.20–8.30; p=0.020).

Conclusions: Elevated TSH levels were independently linked to MetS in postmenopausal women. High normal TSH could help identify women at higher cardio-metabolic risk, and prompt metabolic evaluation.

Keywords: thyroid-stimulating hormone; metabolic syndrome; postmenopausal women; thyroid function; dyslipidemia; central obesity; cardiometabolic risk

INTRODUCTION

Metabolic syndrome (MetS) is a cluster of interrelated cardiometabolic abnormalities characterized by central obesity, elevated blood pressure, impaired glucose regulation, hypertriglyceridemia, and reduced high-density lipoprotein cholesterol (HDL-C). The presence of MetS is associated with a substantially increased risk of type 2 diabetes mellitus, atherosclerotic cardiovascular disease, and premature mortality [1]. Its prevalence increases with advancing age and is particularly high among postmenopausal women, in whom hormonal and metabolic changes may promote the development of multiple cardiovascular risk factors[2,3].

The menopausal transition is accompanied by a marked decline in circulating estrogen levels together with changes in body composition, fat distribution, lipid metabolism, and insulin sensitivity. Postmenopausal women commonly develop increased visceral adiposity, higher triglyceride concentrations, reduced HDL-C, impaired glucose tolerance, and increased blood pressure[4]. These changes contribute to the increased prevalence of MetS after menopause and emphasize the importance of identifying additional endocrine and metabolic factors that may influence cardiometabolic risk in this population[5,6].

Thyroid hormones play a central role in the regulation of basal metabolic rate, thermogenesis, lipid metabolism, glucose utilization, body weight, and cardiovascular function. Overt hypothyroidism is well recognized to be associated with weight gain, dyslipidemia, insulin resistance, and hypertension[7]. However, increasing evidence suggests that more subtle alterations in thyroid function may also be metabolically relevant. Higher serum thyroid-stimulating hormone (TSH) concentrations, even within the conventional euthyroid reference range, have been associated with obesity, elevated triglycerides, reduced HDL-C, insulin resistance, and MetS in several populations[8].

Previous studies have reported a progressive increase in MetS prevalence with increasing TSH concentrations among euthyroid women. Park et al. demonstrated a significant association between TSH levels and MetS in euthyroid postmenopausal women, with women in the highest TSH quartile showing greater metabolic risk compared with those in the lowest quartile[4]. Similar relationships between higher TSH levels and individual metabolic abnormalities have also been observed in other Asian populations. Nevertheless, the association remains controversial because obesity, insulin resistance, age, menopausal duration, and other metabolic factors may themselves influence TSH secretion and thyroid hormone metabolism[5,6].

Data regarding the relationship between TSH levels and MetS among postmenopausal women in Pakistan remain limited. Understanding this association may help identify postmenopausal women who are at increased cardiometabolic risk even in the absence of overt thyroid dysfunction. Therefore, the present study aimed to evaluate the association between serum TSH levels and metabolic syndrome among postmenopausal women attending Sindh Government Hospital Saudabad, Karachi, and the Department of Obstetrics and Gynaecology, Sindh Rangers Hospital, Pakistan[9,10].

MATERIAL AND METHODS

Study Design and Setting: This analytical cross-sectional study was conducted at Sindh Government Hospital Saudabad, Karachi, Pakistan, and the Department of Obstetrics and Gynaecology, Sindh Rangers Hospital, Pakistan, from June 2022 to June 2023. The aim of the study was to assess the relationship of serum thyroid-stimulating hormone (TSH) levels with metabolic syndrome in postmenopausal women.

Study Population and Sampling: One hundred and twenty postmenopausal women were recruited by non-probability consecutive sampling technique. Postmenopause was considered to be the lack of spontaneous menstruation for 12 months or more, for no other physiological or pathological cause. Women with natural menopause aged 45–70 years with written informed consent were included. The required sample size was achieved

Received on 24-11-2023
Accepted on 29-12-2023

from outpatients and gynecology services of the participating hospitals.

Women who had previous diagnosis of overt hypothyroidism or hyperthyroidism, a history of thyroid surgery or radioactive iodine therapy, were currently taking levothyroxine or antithyroid drugs, were menopausal, had hormonal replacement therapy, had active malignancy, severe hepatic or renal disease, acute systemic illness or they were taking drugs known to have significant effects on thyroid function were excluded from the study.

Clinical and Anthropometric Assessment: A structured data collection form was used to document demographic and clinical data. These data comprised age, age at menopause, years since menopause, smoking habits, physical activity, history of hypertension, diabetes mellitus, dyslipidaemia and use of corresponding drugs. A clinical examination was performed for each participant.

Body weight was taken on a calibrated digital weighing scale with the participants in light clothes and without shoes. Body mass index was defined as weight (kg)/height (m2) and height was measured with a stadiometer. Waist circumference was taken at the end of a normal expiration at the level half way between the lower border of the last palpable rib and the upper border of the iliac crest. Blood pressure was taken after 5 minutes of sitting rest with a proper sized cuff. Two readings were taken about five minutes apart and the average of the two readings used for analysis.

Biochemical Assessment: Blood samples were taken from the veins after an 8-hour or more fast. Laboratory tests were blood glucose, total cholesterol, triglyceride, HDL-C, LDL-C, serum TSH and FT4 levels. The concentrations of serum TSH and FT4 were determined by an automated chemiluminescent immunoassay (ICA) following the manufacturer's instructions. Laboratory parameters of fasting plasma glucose and lipid profile were studied in the participating hospital laboratories by standard automated enzymatic method.

Euthyroid status was defined as a serum TSH concentration within the laboratory reference interval (0.40–4.50 mIU/L) and an FT4 concentration within the laboratory reference interval for the primary analysis.

Definition of Metabolic Syndrome: Metabolic syndrome was defined as meeting the harmonized criteria, proposed by the International Diabetes Federation, American Heart Association, and National Heart, Lung, and Blood Institute. A person was considered to have metabolic syndrome if three or more of the five metabolic abnormalities were detected. These were defined as: waist circumference > the appropriate population-specific cut-off; serum triglycerides ≥ 150 mg/dl (or treatment of hypertriglyceridaemia); HDL-C < 50 mg/dl (or treatment for low HDL-C); SBP ≥ 130 mmHg or DBP ≥ 85 mmHg (or current antihypertensive treatment); and fasting plasma glucose ≥ 100 mg/dl (or treatment of hyperglycaemia).

Classifications of TSH levels: Serum TSH levels were divided into quartiles based on their distribution in study population. The participants were divided into 4 groups of similar size, according to their TSH levels, from lowest to highest. The prevalence of metabolic syndrome and its components were assessed according to increasing TSH levels with this approach.

Statistical Analysis: IBM SPSS Statistics was used for data analysis. Normally distributed continuous variables were expressed as mean ± standard deviation and other continuous variables were expressed as median (interquartile range). Categorical data were presented as frequencies and percentages. Independent-samples t-test or Mann-Whitney U test was used to compare continuous variables in women with metabolic syndrome and in women without metabolic syndrome, depending on data distribution, and the chi-square test or Fisher's exact test was used to compare categorical variables.

Pearson's or Spearman's correlation analysis was used to evaluate the correlation between serum TSH concentrations and continuous metabolic parameters. Trend of prevalence of metabolic syndrome with descending order of TSH quartiles was assessed by chi-square test of trend. Binary logistic regression analysis was performed to determine the association between serum TSH and metabolic syndrome. Crude and adjusted odds ratios (OR) with 95% confidence intervals were estimated. To assess for possible confounding factors, age, years since menopause, BMI, FT4 concentration, smoking status and physical activity were included in the multivariable model. Statistically significant p values were set at <0.05 (two sided).

Ethical Considerations: The study was performed in compliance with principles of the Declaration of Helsinki. The study was conducted after receiving ethical clearance from the relevant Institutional Review Board or Ethical Review Committee. All participants provided written informed consent before participation. The identity of the participants and clinical information were kept confidential and used solely for research. The actual ethical approval number is to be inserted when available in the final manuscript.

RESULTS

Participant Characteristics: A total of 120 postmenopausal women were included in the final analysis. The mean age of the population studied was 57.8 ± 6.2 years. Of the 120 women, 59 (49.2%) had metabolic syndrome while 61 (50.8%) were not diagnosed with metabolic syndrome.

The women with metabolic syndrome were significantly older than the women without metabolic syndrome (59.4 ± 6.1 vs. 56.3 ± 5.9 years; p=0.005), and they were older at the time of menopause (8.5 ± 4.8 vs. 6.2 ± 4.1 years; p=0.005). Waist circumference and BMI were significantly higher in women with metabolic syndrome. Also, the metabolic syndrome group had significantly elevated levels of systolic and diastolic BP, FPG, triglycerides and LDL-C, and significantly reduced levels of HDL-C. Further, the serum TSH level was significantly higher in women with metabolic syndrome than in women without metabolic syndrome (2.61 vs. 1.79 mIU/L; p<0.001). Distribution of FT4 did not differ significantly between the two groups (Table 1).

Participants were categorized into four TSH quartiles, with 30 women in each group. The prevalence of metabolic syndrome increased progressively with increasing TSH concentration. Metabolic syndrome was present in 9 women (30.0%) in the lowest TSH quartile, 12 (40.0%) in the second quartile, 16 (53.3%) in the third quartile, and 22 (73.3%) in the highest quartile. This demonstrated a statistically significant increasing trend across TSH categories (p for trend <0.001).

A similar pattern was observed for several individual components of metabolic syndrome. Central obesity increased from 36.7% in the lowest TSH quartile to 76.7% in the highest quartile, while the prevalence of elevated triglycerides increased from 30.0% to 66.7%. Low HDL-C, elevated blood pressure, and elevated fasting plasma glucose also became progressively more frequent with increasing TSH concentrations (Table 2).

There were significant positive correlations between the serum TSH concentration and several adverse metabolic parameters. TSH was positively correlated with waist circumference (r=0.31; p=0.001), BMI (r=0.27; p=0.003), triglyceride concentration (r=0.33; p<0.001), systolic blood pressure (r=0.20; p=0.029), diastolic blood pressure (r=0.21; p=0.021), and fasting plasma glucose (r=0.19; p=0.038).

On the other hand, serum TSH was significantly inversely correlated with HDL-C level (r=-0.29, P=0.001). Triglyceride concentration and waist circumference were the most positively associated with the women (Table 3).

The odds of metabolic syndrome increased with each quartile increment of TSH by binary logistic regression analysis. Compared with women in the lowest TSH quartile, those in the highest quartile had substantially greater unadjusted odds of metabolic syndrome (OR 6.42; 95% CI 2.08–19.76).

Even after adjustment for age, duration since menopause, BMI, FT4 concentration, smoking status, and physical activity,

women in the highest quartile of TSH maintained significantly increased odds of metabolic syndrome when compared with women in the lowest quartile (adjusted OR 3.15; 95% CI 1.20–8.30; p=0.020). These relationships for the second and third quartiles were also reduced upon adjustment and were not statistically significant (Table 4).

When TSH was analyzed as a continuous variable, each 1 mIU/L increase in serum TSH was associated with a 46% increase in the adjusted odds of metabolic syndrome (adjusted OR 1.46; 95% CI 1.10–1.94; p=0.009). Overall, these findings demonstrated a significant positive relationship between increasing serum TSH concentration and the burden of metabolic abnormalities among postmenopausal women.

Table 1: Clinical and biochemical characteristics according to metabolic syndrome status

Variable	Without MetS (n=61)	MetS (n=59)	p value
Age, years	56.3 ± 5.9	59.4 ± 6.1	0.005
Duration since menopause, years	6.2 ± 4.1	8.5 ± 4.8	0.005
BMI, kg/m²	26.7 ± 3.6	30.8 ± 4.1	<0.001
Waist circumference, cm	83.1 ± 8.4	94.0 ± 9.0	<0.001
Systolic blood pressure, mmHg	123.8 ± 13.5	137.6 ± 15.1	<0.001
Diastolic blood pressure, mmHg	77.9 ± 8.0	85.7 ± 8.9	<0.001
Fasting plasma glucose, mg/dL	91.8 ± 11.0	110.2 ± 22.1	<0.001
Triglycerides, mg/dL	123 (96–149)	184 (154–225)	<0.001
HDL-C, mg/dL	52.5 ± 9.5	42.1 ± 8.2	<0.001
LDL-C, mg/dL	115.9 ± 28.6	132.4 ± 31.8	0.003
TSH, mIU/L	1.79 (1.20–2.40)	2.61 (1.91–3.31)	<0.001
FT4, ng/dL	1.15 ± 0.16	1.12 ± 0.17	0.322

Distribution of Metabolic Syndrome Across TSH Quartiles

Table 2: Prevalence of metabolic syndrome and its components across TSH quartiles

Variable	Q1 (n=30)	Q2 (n=30)	Q3 (n=30)	Q4 (n=30)	p for trend
Metabolic syndrome	9 (30.0%)	12 (40.0%)	16 (53.3%)	22 (73.3%)	<0.001
Central obesity	11 (36.7%)	15 (50.0%)	19 (63.3%)	23 (76.7%)	0.001
Elevated triglycerides	9 (30.0%)	12 (40.0%)	16 (53.3%)	20 (66.7%)	0.003
Low HDL-C	11 (36.7%)	14 (46.7%)	18 (60.0%)	21 (70.0%)	0.006
Elevated blood pressure	10 (33.3%)	12 (40.0%)	16 (53.3%)	19 (63.3%)	0.012
Elevated fasting glucose	7 (23.3%)	9 (30.0%)	11 (36.7%)	14 (46.7%)	0.046

Correlation Between TSH and Metabolic Parameters

Table 3: Correlation between serum TSH and metabolic parameters

Parameter	Correlation coefficient (r)	p value
BMI	0.27	0.003
Waist circumference	0.31	0.001
Systolic blood pressure	0.20	0.029
Diastolic blood pressure	0.21	0.021
Fasting plasma glucose	0.19	0.038
Triglycerides	0.33	<0.001
HDL-C	-0.29	0.001
LDL-C	0.18	0.049

Association Between TSH Levels and Metabolic Syndrome

Table 4: Logistic regression analysis of the association between TSH quartiles and metabolic syndrome

TSH quartile	MetS, n/N	Crude OR (95% CI)	Adjusted OR (95% CI)	p value
Q1	9/30	Reference	Reference	—
Q2	12/30	1.56 (0.53–4.53)	1.35 (0.43–4.22)	0.606
Q3	16/30	2.67 (0.92–7.70)	1.88 (0.61–5.79)	0.271
Q4	22/30	6.42 (2.08–19.76)	3.15 (1.20–8.30)	0.020

DISCUSSION

The current study showed that serum thyroid-stimulating hormone (TSH) levels were significantly related to metabolic syndrome (MetS) in postmenopausal women[1]. The median concentrations of TSH were significantly higher in women with MetS than in women without MetS and the prevalence of MetS increased progressively with TSH quartiles. Of interest, there was a significantly higher prevalence of women who met the diagnostic definition of MetS in the highest quartile of TSH than in the lowest quartile (73.3% vs. 30.0%). Despite adjustment for age, duration since menopause, body mass index, FT4, smoking status, and physical activity, women in the highest quartile of TSH levels still had significantly higher odds of MetS[2,3].

The results obtained here were similar to those of Park et al., who showed a progressive rise in the prevalence of MetS in the successive quartiles of TSH levels in euthyroid postmenopausal women4. Their study also indicated that there might be an unfavourable cardiometabolic profile with relatively higher TSH concentrations even within the conventional euthyroid range. Similar correlation of increased TSH levels with metabolic abnormalities has also been observed in other euthyroid female and Asian populations[5,6].

The present study also showed that TSH was positively correlated with BMI, WC, Trig, FPG and BP while it was negatively correlated with HDL-C. Of these variables, the most significant relationships were seen with triglycerides and waist circumference. These observations support the hypothesis that relatively lower thyroid functional activity may contribute particularly to abnormalities in lipid metabolism and central adiposity[7,8].

There are several biological mechanisms that could account for these findings. Thyroid hormones influence basal energy expenditure, thermogenesis, hepatic lipid metabolism, lipolysis, glucose utilization and vascular tone. A relative decrease in thyroid functional state increases resting energy expenditure, lowers lipid clearance, and leads to weight gain and hypertriglyceridemia[9,10]. Reduced thyroid hormone activity may also decrease lipoprotein lipase activity and hepatic LDL-receptor expression, contributing to an adverse lipid profile[11].

Thyroid function also may be bidirectionally linked to central obesity. Adiposity is linked to changes in leptin secretion and inflammatory signals which both have an effect on the activity of the hypothalamic-pituitary-thyroid axis. Consequently, higher TSH concentrations may not only contribute to metabolic disturbances but may also occur as a physiological response to obesity and insulin resistance[12].

The relationship between TSH and FPG seen in the current study may also be due to interaction between thyroid function and insulin sensitivity[13]. Thyroid hormones have effects on glucose production in the liver, uptake of glucose in the peripheral tissues, and insulin-mediated metabolic pathways. Thus, mild decreases in thyroid function could play a role in abnormal glucose metabolism. This interaction may be of special significance in postmenopausal women as estrogen deficiency alone is known to induce visceral adiposity and insulin resistance[14].

The relationship between increased TSH levels and hypertension, observed in this study, is also biologically plausible. Low thyroid hormone levels can lead to increases in systemic vascular resistance, decreased endothelial function, and changes in renal sodium handling. The correlations with systolic and diastolic blood pressure were not as strong as those with triglycerides and waist circumference, but were statistically significant[15,16].

The results that we found are also in line with the evidences that have been found, that subclinical hypothyroidism is associated with MetS. The results of a meta-analysis of observational studies[8] reported by Ding et al. indicated that there is an association between subclinical hypothyroidism and the risk of MetS. These findings suggest that the relationship between thyroid function and metabolic risk may exist across a continuum rather than being restricted to overt thyroid disease[17].

But, some studies have failed to determine TSH as an independent determinant of MetS. Mehran et al. reported that FT4 was more strongly associated with MetS than TSH after adjustment for relevant confounders[9]. Differences in ethnicity, age, menopausal status, obesity prevalence, iodine intake, laboratory

reference ranges, and statistical adjustment may partly explain these inconsistencies[2].

The present study found that the BMI and other confounding factors did not affect the statistically significant relationship between the highest quartile of TSH and MetS. This indicates that obesity might not be the only contributor to the association between TSH and MetS. However, the reduction in the OR on multivariable adjustment suggests that adiposity and other metabolic factors play a significant role in this association[5-10].

In clinical terms, postmenopausal women with TSH levels at the higher end of the normal reference range might be a subpopulation of women who are more likely to have cardiometabolic abnormalities[18] .Thus, in these women, comprehensive assessment of waist circumference, blood pressure, fasting glucose and lipid profile may be particularly relevant. But the current results do not prove that thyroid hormone treatment is helpful in euthyroid women with high-normal TSH levels, and treatment should still be based on known thyroid disease guidelines[19].

There are several strengths in this study such as two hospital recruitment sites, assessment of multiple components of MetS, evaluation of both TSH and FT4 level and multivariable adjustment for important potential confounding factors[20]. But there are a few drawbacks to keep in mind. The cross section designs do not allow causality or temporal relationships to be inferred. The sample size is relatively small, which could restrict the accuracy and validity of the statistical analyses and the generalizability of the results. TSH was only measured once and thyroid autoantibodies, fasting insulin, HOMA-IR, inflammatory markers, dietary iodine intake, and reproductive hormone levels were not evaluated. Additionally, there may be residual confounding due to diet, socioeconomic status, medication use and lifestyle factors[7-12].

Future prospective studies with larger populations and repeated measurements of thyroid function should be conducted to confirm if increased TSH is a predictor of subsequent development of MetS, DM, and/or CVD among postmenopausal women [15-20].

CONCLUSION

In the postmenopausal women, higher serum TSH level was found to be significantly associated with the presence of metabolic syndrome. The proportion of MetS was gradually rising with the quartiles of TSH and women in the highest quartile of TSH had significantly higher adjusted odds of MetS compared with women in the lowest quartile of TSH. Central obesity, higher triglycerides, lower HDL-C, and poor glucose and blood pressure profiles were specifically related to higher TSH concentrations. These results indicate that relatively high TSH levels, even within the euthyroid range, could potentially help identify postmenopausal women at higher risk for cardiometabolic disease. Future studies should confirm whether high-normal TSH levels are causally associated and are clinically important in this population.

Competing Interests: The authors declare that they have no competing interests.

Funding: No external funding was received for this study.

Authors' Contributions: ST and NIR contributed to study conception and design. MI and NG contributed to data collection and clinical assessment. SLP contributed to laboratory evaluation and interpretation of biochemical findings. DES contributed to data analysis and interpretation. ST and DES drafted the manuscript. NIR, MI, NG, and SLP critically reviewed the manuscript for important intellectual content. All authors read and approved the final manuscript.

Acknowledgment: The authors sincerely acknowledge the administrative, clinical, and laboratory staff of Sindh Government Hospital Saudabad, Karachi, and the Department of Obstetrics and Gynaecology, Sindh Rangers Hospital, Pakistan, for their support during data collection and laboratory assessment. The authors also thank all study participants for their valuable cooperation and participation in this research.

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This article may be cited as: Tabassum S., Ranjha N. I., Ilyas M., Gul N., Prithiani S. L., Shehwar D. E, Association Between Thyroid-Stimulating Hormone Levels and Metabolic Syndrome in Postmenopausal Women. Pak J Med Health Sci, 2023; 18(1): 1083-1086.