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Original Article

Relationship Between TSH Level and Cardiometabolic Risk Factors in Overweight and Obese Adolescents

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Abstract ***Purpose:*** To investigate the relationship between TSH levels and cardiometabolic risk factors in overweight/obese adolescents by gender. ***Methods:*** We performed a retrospective cross-sectional analysis of the data from 343 overweight/obese adolescents aged between 11 and 18 years. The degree of obesity was calculated as the body mass index standard deviation score (BMI-SDS). Hypertension, dyslipidaemia, hyperinsulinaemia, hyperglycaemia and insulin resistance were defined as cardiometabolic risk factors. The patients' TSH and free T4 levels were recorded, and the subjects with normal free T4 levels were included. ***Findings:*** A positive correlation was found between TSH level and homeostatic model assessment of insulin resistance (HOMA-IR), insulin and triglyceride levels (p=0.001, 0.001, 0.006, respectively). In the linear regression analysis, in which age, gender and BMI-SDS values were taken as covariates, a 10% increase in the geometric mean of TSH was associated with a 0.13 fold increase in HOMA-IR, and a 10% increase in TSH level was associated with a onefold increase in geometric mean of insulin level (p=0.003, 0.002, respectively), but the relationship between TSH and triglyceride levels disappeared. ***Conclusions:*** TSH levels were found to be related to the glucose metabolism. Further prospective studies are needed to clarify the mechanism of this relationship.

Key words *Adolescents; Cardiometabolic risk factors; Obesity; TSH level*

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Introduction

The frequency of overweight and obesity is gradually increasing worldwide.[1] According to the data of the World Health Organisation, obesity has nearly tripled since 1975.[1] Among overweight or obese children and adolescents, cardiometabolic risk factors, such as hypertension, insulin resistance, dyslipidaemia are more prevalent than those of normal weight peers.[2-5] Also, high serum TSH and normal thyroid hormone concentrations have been reported in obese children and adolescents, and this situation has been referred as "isolated hyperthyrotropinaemia (IH)" when this hormone profile is not accompanied by circulating thyroid antibodies.[6,7] High serum TSH levels were found to be normal after weight loss indicating the association of the presence of high TSH levels in obesity.[6,7] Also, it was ultrasonographically shown that thyroid structure was altered in overweight and obese children, and improved after weight loss.[7] Lundbäck et al found that there was a positive association between TSH levels and degree of obesity in obese children even if the TSH levels were within

the normal range.[8] Still, the reasons for alterations in thyroid hormone levels in obese children remain unclear.[9] Thyroid hormone resistance, mutations in TSH receptor gene, increased leptin levels, alterations in peripheral thyroid hormone deiodinase activity, impaired mitochondrial functions, and various other mechanisms such as iodine deficiency, autoimmune thyroiditis and defects in hypothalamic-pituitary axis might be the causes of increased TSH levels among obese children.[9]

Among overweight/obese children and adolescents, higher serum TSH concentrations were found to be associated with cardiometabolic risk factors, and it is controversial whether the morbidities frequently associated with obesity might be negatively influenced by concommitant presence of hyperthyrotropinemia.[6,8,10-14] The primary aim of this study was to investigate the relationship between thyroid hormones, degree of obesity and cardiometabolic risk factors in overweight and obese Turkish adolescents by gender. The secondary aim of this study was to assess the demographic and cardiometabolic risk factor differences in overweight and obese adolescents with TSH levels within the normal range, in comparison to patients with high TSH levels.

Materials and Methods

We obtained data retrospectively from medical records of overweight or obese adolescents aged between 11 and 18 years who attended Istanbul University-Cerrahpasa Medical Faculty Adolescent outpatient clinic from January 2012 to December 2015 to evaluate the association between TSH level and cardiometabolic risk factors among overweight and obese adolescents. Ethics committee approval was obtained for the study: Cerrahpasa Medical Faculty Deanship Clinical Research Ethics Committee, Date: 20.06.2016, number: 29430533- 604.01.01-225233.

In our country, parents can schedule appointments for their teens via a telephone call or online, and adolescents can be examined, investigated and treated at adolescent outpatient clinics regardless of their complaints. In our adolescent outpatient clinic, we routinely calculate the body mass index (BMI) of patients whatever the reason of admission is, and if we determine that the patient is overweight/obese, we evaluate him/her in terms of cardiometabolic risk factors that might be associated with obesity. The patients who were included in this study were either admitted to our clinic for weight management or for an acute transient health problem, and were found to be overweight/obese during routine evaluation. We used the same methods to grade obesity and to define cardiometabolic risk factors as in our previous study as we described below.[15] Three hundred forty-three overweight or obese adolescents who met the Cole at al's overweight and obesity criteria according to age and gender and who were in puberty, were included in the study.[16] The adolescents who had any chronic disease, who were using any medication that could affect thyroid functions, and whose free T4 levels were not in the normal range, were excluded. The blood samples for thyroid function tests were drawn after the acute illness period passed, in the morning and after 8-12 hours fasting. Age, gender, weight, height, blood pressure, total cholesterol, triglyceride, HDL cholesterol, LDL cholesterol, fasting blood glucose, insulin, TSH, and free T4 levels of the participants were recorded from the patients' files.[16] The normal range of values reported in the kit used (Roche modular system electrochemiluminescence method) for TSH was 0.4-4.2 mIU/L, and TSH levels higher than 4.2 mIU/L were defined as high TSH levels.

Weight status was classified on the basis of the measured height and weight obtained at the time of physical examination, and BMI was calculated using the following formula: BMI=[weight/height^2 (kg/m^2)].[1] According to Cole et al's. criteria, BMI values between the 85th and 95th percentiles were accepted as overweight and BMI values above the 95th percentile were accepted as obese.[16] The degree of obesity was calculated as the body mass index standard deviation score (BMI-SDS) using age and gender specific Turkish BMI percentiles, which were generated by using lambda, mu, sigma (LMS) method, to standardise the degree of obesity.[16,17] The LMS method provides a way for obtaining normalised growth centile standards which simplifies the assessment of growth standards and summarises the data in terms of three smooth age-specific curves called L (lambda), M (mu), and S (sigma).[17]

Hypertension, dyslipidaemia, hyperinsulinaemia, hyperglycaemia and insulin resistance were defined as cardiometabolic risk factors. We used standard cut-off values for levels of fasting blood glucose (>5.6 mmol/L), total cholesterol (≥5.17 mmol/L), HDL cholesterol (<1.03 mmol/L), LDL cholesterol (≥3.36 mmol/L), and triglycerides (≥1.47 mmol/L) to define abnormal values.[18] Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the equation: HOMA-IR=Fasting insulin (μU/mL) x Fasting glucose (mmol/L)/ 22.5.[19,20] Seated blood pressure (BP) was measured by auscultatory method after the participant had been resting

quietly for 10 minutes. We used standardised blood pressure tables in which abnormal BP values were defined as >95th percentile.[21]

Statistical Analysis

The Statistical Package for Social Sciences version 21.0 statistical package was used for statistical analyses. The data were assessed for normality using visual and analytic methods. Continuous variables were defined as mean±SD, and categorical variables were defined as percentages. In the assessment of the relationship between cardiometabolic risk factors and TSH level, Spearman's correlation test was used. A series of linear regressions were conducted to determine whether TSH level significantly contributed to the cardiometabolic risk factors. Sex, age and BMI-SDS were considered as covariates in the aforementioned models. Because insulin and TSH levels did not show a normal distribution, logarithmic transformation of insulin and TSH were included in the search for correlations and linear regression analysis. The UCLA Institute for Digital Research and Education was used in interpreting logarithmic models.[22] Student's *t* test was used in groups with normal distribution and Mann-Whitney *U* test was used in the groups without normal distribution. Comparison between the groups was done using Chi-square test. A *p* value of <0.05 was considered statistically significant.

Results

Among the 343 adolescents with a BMI at the 85th percentile or higher, 25.1% were overweight, and 74.9% were obese. In the study group, the mean age was 14.03±1.76 years, and 59.8% of the subjects were female. The mean BMI value was found to be 30.39±4 kg/m^2 and the BMI-SDS value was found to be 2.4±0.64. In this study sample, 86% of the subjects exhibited normal TSH levels and 14% exhibited high TSH levels.

The correlations between TSH levels and cardiometabolic risk factors are presented in Table 1. Using Spearman correlation test, it was shown that HOMA-IR, insulin and trygliceride levels increased as the TSH level increased (p=0.001, 0.001, 0.006, respectively). No significant correlation was found between TSH and BMI-SDS, total cholesterol, HDL-cholesterol, LDL-cholesterol and fasting blood glucose level. In the correlation analysis in which 295 subjects with normal TSH levels were evaluated, TSH levels were only found to be positively correlated with insulin levels and HOMA-IR (both r, p values=0.161, 0.006). No statistically significant correlation was found between TSH levels within the reference range and BMI-SDS and other cardiometabolic risk factors (data not shown).

The results of the linear regression analysis in which the relationship between the cardiometabolic risk factors and TSH level was evaluated, and sex, age and BMI-SDS

Table 1 Relationship between the cardiometabolic risk factors and TSH level

	TSH (μIU/mL)					
	Whole group		Female		Male	
	r	*p**	*r*	*p**	*r*	*p**
BMI-SDS	0.007	0.891	0.000	0.995	0.107	0.211
SBP (mmHg)	-0.056	0.307	-0.049	0.495	-0.083	0.334
DBP (mmHg)	-0.052	0.344	-0.054	0.453	-0.068	0.430
Total cholesterol (mmol/L)	0.074	0.183	0.088	0.218	0.056	0.530
HDL-cholesterol (mmol/L)	-0.062	0.265	-0.095	0.184	0.052	0.554
LDL-cholesterol (mmol/L)	0.047	0.391	0.076	0.287	-0.026	0.769
Triglyceride (mmol/L)	0.149	**0.006**	0.153	**0.031**	0.112	0.196
Fasting blood glucose (mmol/L)	0.074	0.173	0.057	0.419	0.094	0.273
Insulin (pmol/L)	0.186	**0.001**	0.203	**0.004**	0.168	**0.049**
HOMA-IR	0.190	**0.001**	0.205	**0.004**	0.173	**0.043**

*Spearman's Correlation Test

DBP: diastolic blood pressure; HOMA-IR: homeostatic model assessment of insulin resistance; BMI-SDS: body mass index standard deviation score; SBP: systolic blood pressure

were considered as covariates, are presented in Table 2. In the linear regression model used to determine the contribution of TSH to HOMA-IR, TSH (β=2.93, p=0.004) and BMI-SDS (β=4.63, p=0.0001) were found to be significant contributors to the model (adjusted R^2= 0.080), which meant that a 10% increase in the geometric mean of TSH was associated with a 0.13 fold increase in HOMA-IR, and one unit increase in BMI-SDS was related to an increase of 1.546 units in HOMA-IR. In the linear regression model used to determine the degree of the relationship between the TSH and insulin levels, TSH (β=3.17, p=0.002) and BMI-SDS (β=6.15, p=0.0001) were found to be significant contributors to the model (adjusted R^2=0.122), which meant that a 10% increase in TSH level was associated with a 2% increase in the geometric mean of insulin level, and one unit increase in BMI-SDS was related to a 32% increase in the geometric mean of insulin level. In the linear regression analysis, the relationship between TSH and triglycerides disappeared.

Homeostasis model assessment of insulin resistance and insulin level were lower in the subjects with a TSH level below 4.2 μIU/mL compared to those with a TSH level above 4.2 μIU/mL (p=0.035, 0.043, respectively) (Table 3). No statistically significant difference was found between the subjects with a TSH value within the reference range and the subjects with a TSH value higher than the upper normal limit of the reference range in terms of age, gender, BMI-SDS and other cardiometabolic risk factors.

Discussion

In this study, we aimed to retrospectively investigate the relationship between TSH level, which is commonly found to be increased in obese adolescents, and cardiometabolic risk factors, in a group of overweight and obese adolescents. In our study sample, 86% of the subjects exhibited normal thyroid function and 14% exhibited high TSH levels. The frequency of hyperthyrotropinemia observed in this sample was similar to the frequency found by Souza et al.[10] Souza et al reported that 13.63% of the overweight and obese adolescents had high TSH levels and they used the term "subclinic hypothyroidism (SH)" in the presence of high TSH levels and normal free T4 levels, - though they did not evaluate thyroid antibodies.[10] The consequences of elevated TSH levels in obesity remain unclear, and it was shown that weight loss in obese children was associated with a decrease in TSH levels.[23] The most common thyroid disorder in obese subjects is an increase in TSH and free T3 (fT3) levels, and these changes could be interpreted as a defence mechanism of the body against weight gain. An increase in deiodinase activity due to fat deposition results in elevation of fT3 level by way of conversion of T4 to T3.[7] In our study fT3 level was not measured.

In the current study, no relationship was found between TSH and the degree of obesity, which was in line with some previous studies[12,14] but not with another study.[8] This discrepancy might be due to different sample sizes and different methods used in these studies. It was suggested that TSH level was related to visceral adipose tissue volume in overweight and obese adults.[24,25] Also, it has been reported that weight is not proportional to height squared in adolescence, casting doubt on the accuracy of BMI percentiles, and suggesting that the tri-ponderal mass index (TMI= [weight/height^3 (kg/m^3)]) estimates body fat levels better than BMI-SDS, especially in males.[25] Therefore, we explored if there was any relationship between TSH level and the TMI, but we could also not find any relationship between TSH and TMI.

In our study, it was found that fasting insulin level and insulin resistance increased as TSH increased, but it was

Table 2 Multivariate Linear Regression Analysis [dependent variables were HOMA-IR and insulin; independent variables were age, gender (1=male, 2=female), degree of obesity (BMI-SDS) and TSH in each model]

Variable	HOMA-IR		Insulin (pmol/L)	
	Beta	**Significance**	**Beta**	**Significance**
Gender	-0.622	0.534	-1.13	0.256
Age	-1.571	0.117	-1.36	0.173
BMI-SDS	**4.63**	**0.0001**	**6.15**	**0.0001**
TSH (μIU/mL)	**2.93**	**0.004**	**3.17**	**0.002**

HOMA-IR: homeostatic model assessment of insulin resistance; BMI-SDS: body mass index standard deviation score

difficult to ascertain a casual relationship between TSH and insulin and insulin resistance among obese adolescents. After regression, TSH levels remained significantly associated with fasting insulin level and HOMA-IR independent of age, gender and BMI-SDS. As expected, insulin level and HOMA-IR were found to be lower in the subjects with a TSH level below or equal to 4.2 μIU/mL compared to those with a TSH level above 4.2 μIU/mL. Since thyroid autoantibodies were not evaluated in our study, the subjects with subclinical hypothyroidism or autoimmune thyroiditis could not be excluded, so the subjects with normal TSH levels were evaluated seperately to find out if normal TSH levels were associated with cardiometabolic risk factors. We found that TSH levels were associated with insulin and HOMA-IR even in the presence of normal TSH levels in overweight and obese adolescents in both genders. Similar to our study, Souza et al found a significant correlation between TSH and HOMA-IR in euthyroid overweight and obese adolescents.[10] Radhakishun et al also reported that increasing TSH levels were associated with impaired glucose metabolism in euthyroid obese children and adolescents.[26] Javed et al reported that increasing concentrations of TSH, even in the normal range, were associated with deterioration of insulin sensitivity in nondiabetic obese adolescent males.[12] Nader et al reported that increasing TSH levels were associated with higher fasting insulin and insulin resistance in euthyroid children and adolescents.[13] Hyperinsulinaemia was reported in subclinic hypothyroid subjects and interpreted as an early sign of impairment of glucose metabolism.[10,27] Souza et al showed that the subjects with "SH" had higher waist circumference (WC) when overweight and obese adolescents with "SH" were compared with euthyroid overweight and obese adolescents.[10] They emphasized WC as one of the best predictors of obesity-related comorbidities in adolescents, so they speculated that the correlation of TSH with WC might contribute to the possible relationship between TSH and insulin resistance.[10]

In this study, dyslipidaemia and hypertension among obese adolescents did not seem to be related with high TSH levels. It can be speculated that dyslipidaemia and hypertension are secondary to obesity rather than thyroid dysfunction. Similar to our study, Özer et al reported that there was no relationship between TSH level and blood pressure in Turkish obese adolescents.[14] Ünüvar et al also did not found any significant difference between lipid levels and blood pressure in obese children and adolescents with or without hyperthyrotropinemia.[6] The finding of a lack of association between TSH and lipids is also similar to that

Table 3 The demographic and cardiometabolic risk factor differences of the overweight and obese adolescents with or without high TSH levels

Demographic variables and cardiometabolic risk factors	TSH≤4.2 μIU/L		TSH>4.2 μIU/L		*P*
	Mean	±SD	Mean	±SD	
Age (years)	14.09	1.78	13.06	1.56	0.2*
Gender (female/male)	182/113		23/25		0.81**
BMI-SDS	2.41	0.63	2.3	0.7	0.54*
SBP (mmHg)	116.75	13.56	116.41	15	0.8*
DBP (mmHg)	75	11.7	73.91	10.443	0.39*
Total cholesterol (mmol/L)	4.22	0.83	4.41	0.79	0.96*
HDL-cholesterol (mmol/L)	1.28	0.32	1.31	0.35	0.29*
LDL-cholesterol (mmol/L)	2.52	0.69	2.61	0.68	0.52*
Triglyceride (mmol/L)	1.19	0.63	1.44	0.82	0.11*
Fasting blood glucose (mmol/L)	5.08	0.48	5.21	0.45	0.45*
Insulin (pmol/L)	144.5	97.72	179.66	125.03	0.043***
HOMA-IR	4.76	3.61	6.07	4.5	0.035*

*Student T test, **Chi-square, ***Mann-Whitney U test

DBP: diastolic blood pressure; HOMA-IR: homeostatic model assessment of insulin resistance; BMI-SDS: body mass index standard deviation score; SBP: systolic blood pressure; SD: standard deviation

of reported by Javed et al in their study.[12] In contrast to our study and others' studies, Radetti et al found that TSH elevation in overweight and obese children and adolescents was associated with higher total cholesterol and blood pressure.[6,11,12,14] Further studies are needed to highlight this relationship.

We examined a range of cardiometabolic risk factor variables in a clinical-based population. However, our study has certain methodologic limitations. Firstly, it was a cross-sectional study, which prevented the evaluation of permanent relationships between the variables under investigation, and the cross-sectional design did not allow us to examine the effects of these abnormal values on future morbidity or mortality. Secondly, visceral adipose tissue was not measured, so it was not possible to assess whether TSH level and insulin resistance were associated with visceral adipose tissue. The final limitation was that the TSH levels were checked only once per patient, as TSH levels could vary with time in response to iodine levels.

Conclusion

In conclusion, the finding of a positive association between TSH and insulin resistance is intriguing, though a casual relationship is difficult to assess. In the present study, TSH levels appear to be associated with impaired glucose metabolism in a sample of overweight and obese adolescents. Prospective longitudinal studies should be conducted to protect future adults by way of finding the mechanism of the relationship between thyroid hormones and obesity-associated cardiovascular complications, and to understand whether a need for intervention exists in subjects with high TSH levels.

Ethical Statement

This retrospective study was approved by the Cerrahpaşa Medical Faculty Deanship Clinical Research Ethics Committee, Date: 20.06.2016, number: 29430533-604. 01.01-225233. and strictly followed the institution's ethical guidelines.

Declaration of Interest

The authors declare that they have no conflict of interest.

Informed Consent

Informed consent was not obtained because the study was conducted retrospectively.

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