Introduction

Obesity is an important public health problem widely seen across the world and affecting both developing and developed nations. Figures published by the World Health Organization show that the global prevalence of obesity has risen approximately three-fold in the 45-year-period since 1975 [1]. The condition has been linked to complications such as insulin resistance, hypertension, systemic inflammation, type 2 diabetes mellitus, cardiovascular diseases, and cancer [2]. Obesity is also associated with numerous endocrine abnormalities, including thyroid dysfunction and central obesity. However, it is unclear whether thyroid disorder in obese individuals is a cause of that condition or an effect. Obesity is the most important social epidemic of the 21st century. The combination of increased thyroid pathology and obesity is particularly noteworthy. Obesity is associated with numerous endocrine abnormalities, including thyroid dysfunction and central obesity. However, it is unclear whether thyroid disorder in obese individuals is a cause of that condition or an effect. Thyroid dysfunction is associated with changes in body temperature, basal energy expenditure independently of total and physical activity, body weight, and body composition [3]. Although thyroid functions are generally normal in obese individuals, body mass index (BMI) and thyroid stimulating hormone (TSH) are positively related, and a mild increase in TSH levels has been shown compared with normal weight individuals [4, 5]. Although the reason for high TSH values in obese individuals is uncertain, thyroid hormone resistance, autoimmune thyroiditis, iodine deficiency, TSH gene mutation, functional disorders in the hypothalamus-pituitary-thyroid axis, impaired mitochondrial function, and leptin-mediated pro-thyroid releasing hormone (pro-TRH) production have all been implicated [6, 7]. The hormone leptin also occupies an important place in adipocyte function, TSH, fasting plasma glucose (FPG), insulin, total cholesterol, triglyceride, and HDL results were recorded. The t test, Mann Whitney U test, chi square test, and Pearson and Spearman correlation tests were employed for data analysis. A p value < 0.05 was considered statistically significant. Results. Seventy-three patients with BMI ≥ 30 kg/m² and 27 non-obese controls were included in the study. Fifty-nine of the individuals enrolled were women and 41 were men, with an average age of 36.4 ± 10.4 years. Significant differences were observed in BMI, FPG, HbA1c, HDL, and TSH values between the groups (p < 0.05). TSH was significantly positively correlated with TSH and BMI, HbA1c, insulin and FPG (p < 0.001, r = 0.360; p = 0.031, r = 0.231; p = 0.021, r = 0.231 and p = 0.017, r = 0.237, respectively). Negative correlation was present between TSH and HDL (p = 0.006, r = –0.272). Conclusions. TSH, fasting blood glucose, HbA1c and insulin values were higher in the obese group compared with the normal weight healthy group, while HDL values were lower. Significant positive correlation was determined between TSH and BMI. These findings support the idea that a slight increase in TSH levels may occur in obese individuals.

Keywords: obesity; body mass index; thyroid stimulating hormone

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The association between obesity and thyroid stimulating hormone in adults

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hypertrophy in obese individuals causes hyperleptinemia, and increased leptin results in increased TSH, or hypothyroidism [8]. Several studies have shown that leptin can also affect hypothalamic TRH production [9, 10].

The aim of this study was to elucidate the relationship between obesity and serum plasma TSH levels.

Material and methods

The records of 73 obese patients with BMI ≥ 30 kg/m² among individuals aged 18–65 presenting to the internal diseases clinic in our center over a one-year period (1 December, 2014, to 1 December, 2015) and a 27-member non-obese group with normal BMI were scanned retrospectively. Individuals with BMI values less than 25 kg/m² were regarded as non-obese, while participants with BMI exceeding 30 kg/m² were defined as obese.

The patients were divided into two groups:

- Group 1 — Obese patients (BMI ≥ 30 kg/m², n = 73).
- Group 2 — Non-obese control group (BMI < 25 kg/m², n = 27).

Individuals with acute infectious conditions, malignancies, inflammatory rheumatic diseases, chronic kidney failure, or acute or chronic liver disease, and pregnant women were excluded.

Parameters Used During Evaluation and Follow-Up

Age, sex, anthropometric measurements, TSH, fasting plasma glucose (FPG), insulin, total cholesterol, triglyceride, and HDL results were collected from the patient files in this retrospective study. The files of 73 obese patients with BMI ≥ 30 kg/m² and 27 individuals with normal BMI were examined retrospectively. All cases’ biochemical parameters (TSH, FPG, insulin, HbA1c, and lipid profile) were recorded. Insulin resistance (IR) was calculated with the Homeostasis Model Assessment (HOMA) index.

HOMA was determined by applying the formula fasting insulin value (µU/mL) x fasting glucose value (mg/dL)/405.

Cases’ demographic characteristics, height, and body weight were recorded.

BMI was calculated using the formula body weight (kg)/height² (m²).

Statistical analysis

Data analysis was performed on IBM SPSS software version 22.0 for Windows (Statistical Package for the Social Sciences Inc., Chicago, IL, USA). Continuous variables were expressed as mean plus standard deviation (SD), and categorical variables as frequency (%). The distributions of the continuous variables were examined by Kolmogorov Smirnov test. The t test, Mann Whitney U test, and Pearson and Spearman correlation tests were applied for data analysis, and the chi square test was used to compare categorical data. P values < 0.05 were considered statistically significant.

Ethical considerations

Ethical approval for the study was obtained from the Gaziantep University Faculty of Medicine Ethics Committee, Turkey (no. 29.11.2015/330). All participants were briefed about the study and gave informed written consent to participate.

Results

Seventy-three patients with BMI values exceeding ≥ 30 kg/m² and 27 non-obese controls were enrolled in this research. All patients’ demographic and laboratory characteristics are shown in Table 1. There was no difference between the groups in terms of mean age (p = 0.188). The groups were also similar in terms of gender distributions (p = 0/580).

However, significant variations were observed between the groups in terms of TSH, BMI, FPG, insulin, HbA1c and HDL levels (p < 0.05). Obese group TSH, FPG, HbA1c and insulin were higher than those of the normal weight group, while HDL values were lower. No significant difference was observed in total cholesterol or triglyceride values between the study groups (p = 0.652 and p = 0.051, respectively).

Comparison of TSH values in the obese and non-obese groups revealed significant positive correlation between TSH and BMI, HbA1c, insulin and FPG in both groups (p < 0.001, r = 0.360, p = 0.031, r = 0.231, p = 0.021, r = 0.231, and p = 0.017, r = 0.237). Significant negative correlation was observed between TSH and HDL (p = 0.006 r = −0.272). Correlation of TSH levels with other variables are shown in Table 2.

Discussion

This study investigated the relationships between TSH, BMI, FPG, HbA1c, total cholesterol, triglyceride, and HDL in obese patients and a non-obese control group. BMI, a practical, inexpensive and easily applied parameter with a high level of accuracy generally recognized in the measurement of obesity, and known to exhibit good correlation with body fat percentage, was employed in this study [11]. A significant difference was observed in TSH levels between the obese patients and the non-obese control group. Additionally, significant positive correlation was determined between TSH and BMI. A similar study evaluated TSH levels in 87 metabolically stable euthyroid women divided into two groups, with BMI above or below 40 kg/m² at physical examination. TSH levels were higher in the morbidly obese group, and positive correlation was determined between BMI and TSH [12]. In another study involving 165 obese and 116 normal weight patients, TSH levels were also higher in the obese group [13]. Knudsen et al. also determined positive correlation between TSH and BMI, and suggested that a high prevalence of obesity in a population might be associated with increased serum TSH levels [14].

Thyroid hormones represent a particular factor of interest in the development and treatment of obesity. The fact that thyroid hormones play a regulatory role in thermogenesis constitutes a potential factor in the development of obesity. Although the reason for TSH elevation in the obese is unclear, it has been suggested that, similarly to insulin resistance, this may be associated with TSH resistance [6]. In addition, parallel weight gain has been observed if elevation in TSH in obese patients persists over a five-year period. Mild TSH elevation is observed in approximately 25 % of obese individuals (generally below 10 IU/l) [15]. There is a pituitary adaptation mechanism that leads to high TSH levels, particularly in morbid obesity, characterized by rapid weight gain [16]. Studies investigating the association be-
The principal limitations of our study are its retrospective design and single-center nature. However, it yields important findings concerning the relationship between obesity and TSH levels, a very widespread health problem. We think that further more extensive studies are now needed to elucidate the relationship between TSH level and obesity. Patients must be screened in terms of total cholesterol and triglyceride values.

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The authors have carefully considered ethical issues (including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc.).

Notes: * — Pearson’s correlation test; ** — Spearman’s correlation test.

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Зв’язок між ожирінням та рівнем тиреотропного гормону в дорослих

Резюме. Актуальність. Ожиріння — найважливіша соціальна епідемія XXI століття. Особливо заслуговує на увагу часте поєднання патології щитоподібної залози (ЩЗ) та ожиріння. Ожиріння пов’язане з численними ендокринними патологіями, які не страждають від ожиріння. З числа обстежених було 59 жінок і 41 чоловік, середній вік яких становив 36,4 ± 10,4 року.

Метою дослідження було з’ясувати взаємозв’язок між ожирінням та рівнем тиреотропного гормону (ТТГ) у плазмі крові.

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Оригінальні дослідження /Original Researches/

Звернення до редакції

В дослідженні були включeni 73 пацієнти з IMT ≥ 30 kg/m² та 27 осіб, які не страждають від ожиріння. З числа обстежених було 59 жінок і 41 чоловік, середній вік яких становив 36,4 ± 10,4 року.

Висновки. Значення ТТГ вірогідно позитивно корелювало із показниками ТТГ та інсуліну, HbA1c, ХС ЛПВЩ та ТТГ між групами (р < 0,05).

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Взаимосвязь между ожирением и уровнем тиреотропного гормона у взрослых

Резюме. Актуальность. Ожирение — серьезная социальная эпидемия XXI века. Особого внимания заслуживает частое сочетание патологии щитовидной железы (ЩЖ) и ожирения. Ожирение связано с многочисленными эндокринными нарушениями, включая дисфункцию ЩЖ и центральное ожирение. Однако неясно, является ли заболевание ЩЖ у людей с ожирением причиной или следствием этого состояния. Целью исследования было выяснить взаимосвязь между ожирением и уровнем тиреотропного гормона (ТТГ) в плазме крови.

Материалы и методы. Под наблюдением находились 73 пациента с ожирением (индекс массы тела (ИМТ) ≥ 30 кг/м²) в возрасте 18–65 лет и группа из 27 человек без ожирения с нормальным ИМТ. Ретроспективно регистрировали возраст, пол, антропометрические измерения, уровень ТТГ, глюкозы в плазме натощак (ГПН), инсулина, HbA1c, ХС ЛПВП и ТТГ между группами (p < 0,05). ТТГ достоверно положительно коррелировал с ТТГ и ИМТ, HbA1c, инсулином и ГПН (p < 0,001, r = 0,360; p = 0,031, r = 0,231; p = 0,021, r = 0,231 и p = 0,017, r = 0,237 соответственно). Отрицательная корреляция наблюдалась между ТТГ и ХС ЛПВП (p = 0,006, r = −0,272). Выводы. Значения ТТГ, глюкозы в крови натощак, HbA1c и инсулина были выше в группе с ожирением по сравнению с группой здоровых людей с нормальным весом, в то время как значения ХС ЛПВП были ниже. Определена достоверная положительная корреляция между ТТГ и ИМТ. Эти данные подтверждают мнение о том, что у людей с ожирением может наблюдаться небольшое повышение уровня ТТГ.

Ключевые слова: ожирение; индекс массы тела; тиреотропный гормон