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Research Article

Fasting Blood Glucose Levels and Lipid Profile in Patients with Thyroid Dysfunction

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Abstract: The function of the thyroid gland is to keep the oxidative metabolism of body tissues at normal levels. The most important effect of thyroid hormones is to increase the metabolic rate and oxygen consumption of all tissues. In this study, we aimed to determine and compare the fasting blood glucose (FBG) levels, lipids and body mass index (BMI) in different thyroid hormone level states. The study included 32 thyrotoxicosis, 32 subclinical hyperthyroidism, 31 hypothyroidism, 34 subclinical hypothyroidism patients and 31 healthy volunteers as control group. In addition to routine blood tests, free T3 (FT3), free T4 (FT4), Thyroid Stimulating Hormone (TSH), FBG and lipid parameters were assessed and BMI was recorded for each patient. Average FBG level was the lowest in hypothyroid group, the highest in thyrotoxic group, and there was statistically significant difference between these two groups ($p = 0.005$). There was a negative correlation between FBG and TSH ($p = 0.03$). Total cholesterol and LDL levels of hypothyroid patients were significantly higher than other groups ($p< 0.05$). In accordance with literature, we determined that thyroid hormones apparently affect glucose and lipid metabolism.
Keywords: Thyroid dysfunction, Fasting plasma glucose, Lipid profile.

INTRODUCTION

The function of the thyroid gland in general, is to keep the oxidative metabolism of body tissues at normal levels. Thyroid hormones are also necessary for normal growth. The most important effect of thyroid hormones is to increase the metabolic rate and oxygen consumption of all tissues of the body [1].

Thyroid hormones stimulates all stages of carbohydrate metabolism. The primary effects are; increase in the absorption of glucose from the gut, increased uptake of glucose by cells, increase in glycolysis and gluconeogenesis. Thyroid hormones increase the production of glucose from non-carbohydrate compounds and worsen the clinical table of diabetes mellitus [2]. Thyroid hormones also affect all stages of lipid metabolism. They increase the degradation- fragmentation of lipids, enhance lipolysis in adipose tissue. Increased levels of thyroid hormones increase plasma free fatty acids, however decrease cholesterol, phospholipid and triglyceride levels [3].

In this study, we aimed to evaluate FBG levels and lipid profile in states of different thyroid hormone levels.

MATERIALS AND METHODS

This study was conducted after obtaining ethics committee approval and written informed consents of patients. Patients were evaluated in five groups: 1) Control group: Patients with normal blood thyroid hormone and TSH levels (n= 31); 2) Thyrotoxicosis group: Patients with increased blood thyroid hormones and suppressed TSH levels (n= 32); 3) Subclinical hyperthyroidism: Patients with normal FT3, FT4 and suppressed TSH levels (n= 32); 4) Hypothyroidism group: Patients with elevated TSH and low FT3, FT4 levels (n= 31); 5) Subclinical hypothyroidism: Patients with normal FT3, FT4 and elevated TSH levels (n= 34).

Patients with malignancies, infections, systemic diseases such as diabetes, under medication with anti-inflammatory and/ or antioxidant therapy were excluded from the study. BMI of all cases were recorded. Venous blood samples were taken from all patients after 12 hours of fasting for biochemical analysis. Glucose, total cholesterol, LDL cholesterol, HDL cholesterol, very low density lipoprotein (VLDL) cholesterol, triglycerides, urea and creatinine were analyzed with Olympus AU 600 autoanalyzer; CELL-DYN 3700 device was usedfor complete blood count;

thyroid function tests (free T3, free T4 and TSH) were determined by chemiluminescence method with Immulite 2000 device.

The data obtained from the study were presented as mean ± standard deviation. SPSS 12.0 Windows package programme was used for statistical analysis. The differences of parametric data between groups were evaluated by ANOVA and post hoc Tukey test; the differences of categorical data were analyzed by chi-square test. The relationship between the parameters were analzed by Pearson correlation analysis method. $p < 0.05$ was considered significant.

RESULTS

In terms of demographic characteristics, age and BMI, between the groups, there was not statistically significant difference ($p > 0.05$) (Table 1).

Total cholesterol and LDL cholesterol levels of hypothyroid patients were higher than thyrotoxic, control and subclinical hyperthyroidism and subclinical hypothyroidism groups; but the only significant difference was between hypothyroidism and thyrotoxicosis groups ($p < 0.05$). The average triglyceride value of hypothyroid patients was lower than the control group ($p = 1.0$), and higher than thyrotoxic patients ($p = 0.2$). Total cholesterol and LDL cholesterol levels of thyrotoxic group were found to be lower, but on the other hand mean triglyceride levels were higher in comparison with the control group ($p < 0.05$, $p = 0.15$ respectively). Mean HDL cholesterol levels were lower in hypothyroid group than control group, but without statistical significance ($p > 0.05$). HDL cholesterol levels of thyrotoxic group was higher than hypothyroid patients and the control group, but without statistical significance ($p > 0.05$). In hypothyroidism group, there was a positive correlation between TSH and total cholesterol and LDL cholesterol ($p < 0.05$) and also between BMI and total cholesterol, LDL cholesterol and triglyceride levels ($p < 0.05$). Mean FBG level was the lowest in hypothyroid patients, the highest in thyrotoxic group, and the statistically significant difference was only between the thyrotoxicosis and hypothyroidism groups ($p = 0.005$). There was a negative correlation between FBG and TSH ($p = 0.03$).

Table-1. Demographic characteristics and laboratory values of the study groups.

	Control (n=31)	**Thyrotoxicosis (n=32)**	**Subclinical hyperthyroidism (n=32)**	**Hypothyroidism (n=31)**	**Subclinical hypothyroidism (n=34)**
Age (year)	46.6 ± 14.8	48.4 ± 18.2	50.3 ± 15.5	46.3 ± 16.9	44.7 ± 13.4
BMI(kg/m^2)	25.5 ± 3.8	24.9 ± 6.8	25.5 ± 4.6	27.6 ± 5.5	26.6 ± 5.3
Total cholesterol (mg/dl)	203.3±47	161.8±42	184.7±44	208.9±43	203.2±49
LDL(mg/dl)	134.0±37	104.4±31	122.0±33	139.5±39	137.7±36
HDL(mg/dl)	50.5±8	52.0±10	46.4±15	49.9±14	48.7±12
Triglyceride (mg/dl)	159.5±100	105.6±53	144.2±116	156.2±66	142.6±86
FBG (mg/dl)	87.4±10	94.9±12	88.0±12	84.3±15	89.3±8

DISCUSSION

Thyroid dysfunction is the term used to describe the various thyroid diseases. In the follow-up and treatment of these diseases; FT3, FT4 and TSH levels are used as follow-up parameters. According to the levels of these three hormones; hyperthyroidism, hypothyroidism, subclinical hyperthyroidism, subclinical hypothyroidism and euthyroidism may be diagnosed [4].

Weight loss, decrease in muscle and fat mass, reduction of fat storages and disorders of some serum lipid parameters are typical symptoms of thyrotoxicosis [5,6]. In hypothyroidism; oxygen consumption and basal metabolic rate and lipolysis decrease, serum triglyceride and cholesterol levels increase. In hypothyroidism, body weight generally increase, however in thyrotoxicosis weight loss is detected in the majority of patients [7]. In our study, although there was not a statistically significant difference between groups in terms of BMI, but it was highest in the hypothyroid group and the lowest in the thyrotoxicosis group, in accordance with the literature.

In thyrotoxicosis, insulin resistance exists with increased insulin clearance and compensatory insulin secretion. Thyroid hormone excess increases the basal insulin stimulated glucose utilization especially in the skeletal muscle tissue. In experimental studies, it has been demonstrated that expression of the main muscular glucose transporter protein GLUT-4 and its mRNA increase with thyroid hormones and has been shown to decrease in hypothyroidism. In an animal model of hyperthyroidism with L-thyroxine administration, it has been shown that volume of beta cells in the pancreatic islets reduced due to apoptosis [8]. This condition is called as "thyroid diabetes". In patients with thyrotoxicosis, rapid gastric emptying and glucose absorption is thought to cause postprandial hyperglycemia [9]. In our study, FBG was determined the highest in the thyrotoxicosis group and the lowest in the hypothyroid group in accordance with the literature,

and the difference between these groups were statistically significant (p= 0.005). There was a negative correlation between FBG and TSH (p= 0.03).

Relationship between insulin resistance and hypothyroidism is less determined than those in thyrotoxicosis. Different results were obtained in few studies on this subject. In an experimental model of hypothyroidism induced with propylthiouracil, there was no difference in terms of basal glucose and insulin [10]. Compared to the control group, in subclinical hypothyroid group fasting insulin levels were high, but HOMA-IR values were similar, but another study has suggested that insulin sensitivity decreased in hypothyroidism [11,12]. In human hypothyroidism, glucose utilization was reduced after intravenous glucose tolerance test. As mentioned, different results about the relationship between insulin resistance and hypothyroidism have been obtained. In hypothyroid patients, low FBG may be attributed to decreased metabolic rate.

Thyroid hormones stimulate both synthesis of fatty acids (lipogenesis) and the breakdown of lipids (lipolysis and fatty acid oxidation). Mainly T3 increases lipogenesis in the liver, white adipose tissue, kidney and heart. This effect is due to increased transcription of lipogenic enzymes. Serum total cholesterol and LDL cholesterol levels are generally high in hypothyroidism, however triglyceride and medium density lipoproteins are found high in some patients with hypothyroidism [13]. Findings of our study were also compatible with these literature data. Mean triglyceride levels of hypothyroid group were lower than control group, and higher than thyrotoxic group, but without statistical significance. In hypothyroid group, there were positive correlations between TSH and both total cholesterol and LDL ($p < 0.05$).The effects of hyperthyroidism on plasma lipids are contrast to hypothyroidism. Hyperthyroidism increases the use of fat as an energy source. The sources of this fat are increased synthesis in the liver and the hydrolysis of triglycerides in white adipose tissue. Thyroid hormones also increase lipolysis by stimulation of lipase enzyme in adipose tissue indirectly, due to increased catecholamines. In hyperthyroidism, serum total cholesterol, LDL cholesterol and HDL cholesterol tend to decrease [14]. In our study, mean total cholesterol and LDL cholesterol levels were found to be lower in the thyrotoxicosis group in comparison with the control group ($p < 0.05$). Both triglyceride and HDL cholesterol levels were higher in thyrotoxicosis group than other groups, but without statistically significance.

CONCLUSION

In this study; in accordance with literature, we determined that thyroid hormones apparently affect glucose and lipid metabolism of the body.

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