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The impact of weight loss on thyroid autoimmunity - Weight loss decreases thyroid peroxidase antibody levels: a retrospective cohort study

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ABSTRACT

Objectives: Within the last two decades, an increase has been seen both in autoimmune diseases and obesity, therefore, the correlation between obesity and autoimmunity has been questioned and many studies have been conducted on this issue. Based on this relationship, we aimed to determine whether the weight loss affects the thyroid peroxidase (TPO) antibody levels of obese individuals with thyroid autoimmunity or not.

Methods: The patients who were aged over 18 years, had a Body Mass Index (BMI) ≥ 30 Kg/m^2 and TPO antibody ≥ 5.60 IU/mL were included in the study. The primary endpoint was the change in TPO antibody levels of the patients at the end of the sixth month of the follow-up. The correlations of TPO antibody levels with anthropometric and laboratory measurements were evaluated.

Results: At the end of the sixth month of follow-up of the patients, TPO antibody levels decreased after weight loss ($p < 0.001$). No significant correlations were found between the differences in weight, fat mass, muscle mass and TPO antibody levels ($p = 0.171$; $p = 0.656$; $p = 0.939$).

Conclusions: Weight loss caused a decrease in the levels of TPO antibody levels in the obese individuals having thyroid autoimmunity pointing that weight loss might be useful to stop the progression or lead to regression of the disease.

Keywords: autoimmunity, autoantibodies, obesity, thyroiditis, thyroid gland, weight loss

Obesity is defined as the accumulation of fat in the body so as to cause illness and is an increasing public health problem since it leads to hypertension, dyslipidemia, cardiovascular diseases, type 2 diabetes and some cancer types [1]. It is known that genetic factors as well as lifestyle and environmental factors play a role in the etiology of obesity. Thyroid autoimmunity is a disease characterized by the formation of special antibodies which target the thyroid gland [2, 3].

Thyroid peroxidase (TPO) is a molecule, which catalyzes very important reactions such as the activation of iodine, iodination of tyrosine residues and coupling of iodinated tyrosine [4]. Thyroid peroxidase antibody positivity is seen in 90% of patients with patients having thyroid autoimmunity and a better correlation has been found between histological findings of thyroiditis and TPO antibody compared to Thyroglobulin (Tg) antibody [5, 6]. Genetic and environmental factors have also been demonstrated in the etiology of thyroid autoimmunity. The environmental factors triggering thyroid autoimmunity include some nutrients (iodine, selenium, vitamin B12, vitamin D

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etc.), pesticides, radiation, some medications (interferonalpha and gamma, Tumor necrosis factor-alpha), pregnancy, infection, stress, smoking and obesity [7].

Within the last two decades, an increase has been seen both in immune-mediated diseases and obesity, especially in the industrialized Western countries [8, 9]. Therefore, the correlation between obesity and autoimmunity has been questioned and many studies have been conducted on this issue [8, 10, 11]. It has been shown that white adipose tissue in obesity is not only an organ where the energy is stored, but also is an endocrine organ, which secretes cytokines that are named adipokines and have proinflammatory activities. It has been demonstrated that cytokines secreted from the white adipose tissue cause inflammation, and especially leptin among adipokines leads to proinflammatory effects by activating T helper (Th1) cells and suppressing T regulatory (Treg) cells. Immune-inflammatory response is exaggerated by increased leptin levels in persons with obesity, causing autoimmune response in people with predisposition. Conversely, decreased leptin levels can cause immune suppression [7, 12, 13]. Therefore, adipokines are thought to play a key role in the interaction between adipose tissue and immune system and in the relationship between obesity, autoimmune and inflammatory diseases [8].

In the result of these studies, the increasing prevalence of obesity and autoimmune diseases has been linked to the relationship between adipokines and the immune system; it has been suggested that complex immunological alterations yield to autoimmune reactions [1, 7, 14]. Based on this relationship, in this study, we aimed to determine whether weight loss of the obese patients with thyroid autoimmunity, who were followed up at an obesity outpatient clinic, caused a difference in the levels of TPO antibodies affecting the course of thyroid autoimmunity.

METHODS

Study Design

Data of this retrospective cohort study were obtained from registries of the patients who consecutively admitted to the the Istanbul Medeniyet University, Göztepe Training and Research Hospital, Obesity Outpatient Clinic for the purpose of losing weight between January and July 2016. The primary endpoint was the change in TPO antibody levels of the patients at the end of the 6th month of follow-up.

The study was performed in accordance with the Declaration of Helsinki and was approved by Istanbul Medeniyet University Göztepe Research and Training Hospital Ethical Committee (2019/0187).

Participants and Data Sources

The inclusion criteria were being aged over 18 years, having a body mass index (BMI) ≥ 30 kg/m^2, a TPO antibody ≥ 5.60 IU/mL and having thyroid autoimmunity according to their TPO antibody levels. The patients having BMI < 30 kg/m^2, TPO antibody < 5.60 IU/m L and patients on antithyroid drugs were excluded. Age, gender, the drugs used, weight, height, BMI, co-morbid diseases, fat mass, fat-free mass, muscle mass and among the laboratory measurements glucose, Hemoglobin A1c (HbA1c), insulin, Alanine transaminase (ALT), Aspartate transaminase (AST), thyroid-stimulating hormone (TSH), TPO antibody, triglyceride (TG), total cholesterol (TC), low-density lipoprotein (LDL) and high-density lipoprotein (HDL) values were recorded from the files of 260 patients followed-up in the obesity outpatient clinic between January 2016 and July 2016, who met the inclusion criteria. The participants were followed-up every 15 days for the first three months and then monthly with dietary counseling sessions and were prescribed a reduced−calorie and balanced diet (50%-55% of energy as carbohydrate, 30% of energy as fat, and 15%-20% of energy as protein) of self−-prepared foods to achieve weight loss. The correlations of TPO antibody levels with anthropometric and laboratory measurements were evaluated. The weight, BMI, fat mass, fat-free mass, muscle mass and TPO antibody levels of 78 patients who attended visits regularly, lost any amount of weight at the end of the 6th month and whom TPO antibody values were measured (Fig. 1) were recorded from their files. The effect of weight loss on TPO antibody levels was evaluated at the end of the 6th month. Seven of 78 patients were started on levothyroxine therapy during follow-up The levothyroxine doses used were as follows; 4 patients 25 mg, 1 patient 50 mg, 1 patient 75 mg, 1 patient 100 mg levothyroxine.

Anthropometric Measurements

The height of each patient was measured with a

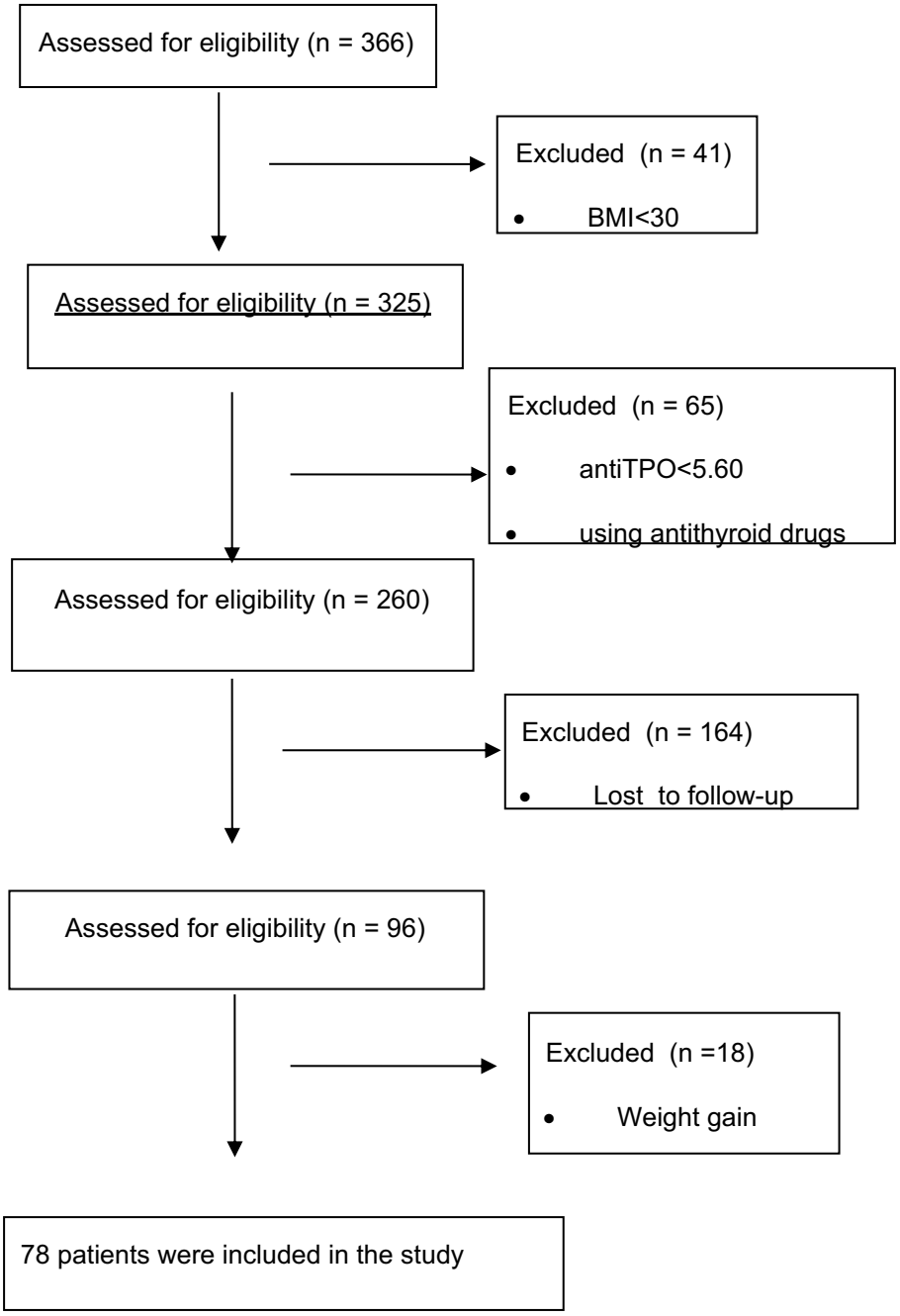

Fig. 1. Flowchart of inclusion/exclusion of participants.

stadiometer (SECA) without shoes and as standing. Weight, fat mass, fat-free mass and muscle mass of each patient was measured with bio-impedance analysis device (TANITA MC 780-MA, Tokyo, Japan). BMI was calculated with the quetelete index (kg/m^2).

Laboratory Measurements

Blood samples were collected following a fasting period of 8 to 12 hours. Fasting blood glucose, TG, TC, LDL, HDL, ALT and AST levels were studied with Roche Cobas 8000 analyzer, while insulin was measured with Beckman Gulter Unicel Dx1 800 and HbA1c using Primus MRDV with HPLC technique. Serum reference ranges were accepted as 0.35-4.49 mIU/L for TSH and a cut-off value of 5.60 IU/mL was taken for TPO antibody and these values were determined using the chemiluminescence (ICMA) method with Architect I2000SR. Patients with a TSH between 0.35-4.49 mIU/L were considered to be euthyroid, 4.50-10.0 IU/mL as subclinical thyroid and > 10.0

IU/mL as overt hypothyroid. Patients with an antiTPO ≥ 5.60 IU/mL were accepted to have thyroid autoimmunity.

Statistical Analysis

Data analyses were performed with the statistical software SPSS for IBM, version 25.0 (SPSS, Inc., Chicago, IL). Normally distributed data were shown as mean ± SD and the data that were not normally distributed were presented as median, minimum and maximum values. Significant differences of normally distributed data were assessed using a t-test and significant difference of not normally distributed data were analyzed using the Mann-Whitney U test. Categorical data were expressed as percentages. Pearson correlation test was applied to test if there is a correlation between weight loss and the decrease in TPO antibodies. A paired two sample test were applied to analyze the effect of weight loss on TPO antibodies. *P* value < 0.05 was statistically significant.

RESULTS

Baseline characteristics of the initial and final sample included in the study are given in Table 1. Mean age of the participants was 49.23 ± 12.49 years. Because most patients presenting to our obesity clinic were women, 90.8% of the participants were female patients. The mean BMI was found as 37.19 ± 5.19 kg/m2, mean fat mass as 38.21 ± 9.78kg, mean fat free mass as 57.55 ± 8.16 kg and mean muscle mass as 54.23 ± 8.16 kg. Median levels of TPO antibody was found as 169.55 (5.78-1000). The characteristics of the initial and the final sample was similar in terms of age (p = 0,08), gender (p = 0.09), weight (p = 0.64), BMI (p = 0.89), fat mass (p = 0.25), fat free mass (p = 0.56), muscle mass (p = 0.85) and TPO antibody (p = 0.54)

Table 1. The baseline characteristics of initial and final samples

	Initial sample (n = 260)		Final Sampes (n = 78)		
	Mean ± SD	Median (Min-Max)	Mean ± SD	Median (Min-Max)	*p* value
Age (years)	49.23 ± 12.49	52(18-78)	51.71 ± 11,26	53,5 (23-78)	0.08
Gender					**0.009**
Female n (%)	236 (90,8)		76 (96.2)		
Male n (%)	24 (9.2)		2 (3.8)		
Weight (kg)	96.26 ± 15.57	94.3 (70.4-149.2)	94.98 ± 15.97	92.98 (70.5-45.8)	0.64
BMI (kg/m^2)	37.19 ± 5.19	36.4 (30-57)	37.01 ± 5.92	35.7 (30-7)	0.89
Fat mass (kg)	38.21 ± 9.78	37 (22.5-80.3)	38.75 ± 10.64	36.5 (23.1-67.6)	0.25
Fat-free mass (kg)	57.55±8.16	56.25 (37.6-86)	56.9 ± 8.0	54.85 (43.6-81.5)	0.56
Muscle mass (kg)	54.23 ± 8.16	53.1 (27.7-91.8)	52.98 ± 8.16	51.85 (27.7-77.5)	0.85
AntiTPO (IU/mL)	315.18 ± 343.71	169.55 (5.78-1000)	321.70 ± 346.95	202 (5.85-1000)	0.54
TG (mg/dl)	153.35 ± 84.24	136.5 (42-779)			
TC (mg/dl)	212.03 ± 42.86	213 (49-322)			
LDL-C (mg/dl)	136.12 ± 36.28	133 (38-231)			
HDL-C (mg/dl)	49.1 ± 15.42	48 (25-188)			
TSH (mIU/l)	3.25 ± 4.11	2.17 (0.35-39.85)			
Glucose (mg/dl)	109.95 ± 43.54	99 (82-439)			
HbA1c (%)	6.47 ± 3.86	5.8 (4.8-14.6)			
Insulin(mIU/l)	12,83 ± 6.62	11.2 (3-45)			
ALT (IU/l)	26.15 ± 16.56	21 (6-103)			
AST (IU/l)	21.93 ± 10.05	19 (9-70)			

BMI = body mass index, Anti TPO = Thyroid peroxidase antibody, TG = triglyceride, TC = total cholesterol, LDL-C = low density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, TSH = thyroid stimulating hormonel, T4 = thyroxin, HbA1c = glycated hemoglobin, ALT = Alanin transaminase, AST = Aspartat transaminase

(Table 1).

Metabolic syndrome was found in 52.04%, hypertension in 32.7%, diabetes in 15.9%, depression in 9.09%, chronic heart disease in 9.09%, chronic obstructive pulmonary disease (COPD) in 4.1%, and obstructive sleep apnea syndrome (OSAS) in 2% of the participants. 82.6% (n = 226) of the participants were in euthyroid, 12.8% (n = 25) of them were in subclinical hypothyroid and 4.6% (n = 9) of the patients were in an overt hypothyroid state (Table 2).

No significant correlation was found between age, weight, fat mass, fat free mass, muscle mass, and biochemical parameters, and TPO antibody levels of the patients. There was a weak correlation between TPO antibody and BMI values (p = 0.042) (Table 2). The correlation between TPO antibody and BMI values was disappeared after adjusting for age, gender and TSH values (p = 0.094). When the correlation between TPO antibody levels and BMI was analyzed separately, no correlations were detected among the patients in euthyroid, subclinical hypothyroid and overt hypothyroid state (Table 2).

After adjusted for age, smoking, menopause and TSH; no significant correlation was found between TPO antibody and BMI and HbA1c values in female patients (p = 0.84, p = 0.88). When both genders were evaluated and the values were adjusted for age, smoking and TSH values, no significant correlation was found between TPO and BMI and HbA1c values (p = 0.932, p = 0.879).

The characteristics of the final sample including 78 patients at the baseline and 6th month as well as the difference of the characteristics between the baseline and 6th month is shown in Table 3. The median TSH levels of the participants at baseline were 2.37 (0.00-39.85). 78.4% (n = 58) of the participants were in euthyroid, 18.9% (n = 14) of them were in subclinical hypothyroid and 2.7% (n = 2) of the patients were in overt hypothyroid state.

The effect of weight loss on TPO antibody in the final sample that lost weight (n = 78) was the primary endpoint of the study and it was found that TPO anti-

Table 2. Correlations of baseline TPO antibody levels with anthropometric and biochemical parameters

	TPO antibody							
	Whole population (n = 260)		Euthyroid (n = 226)		Subclinical hypothyroid (n = 25)		Overt hypothyroid (n = 9)	
	r	*p* value	r	*p* value	r	*p* value	r	*p* value
Age (years)	-.069	.268	-.154	.052	.304	.134	-.013	.974
BMI (kg/m^2)	**.126**	**.042**	.037	.645	.336	.100	.448	.226
Weight (kg)	.042	.497	-.037	.639	.262	.206	.492	.178
TC (mg/dl)	.004	.950	.022	.790	-.009	.966	-.104	.790
LDL (mg/dl)	-.010	.883	.012	.883	.004	.984	-.122	.755
HDL (mg/dl)	.014	.847	.085	.301	-.242	.254	-.509	.162
TG (mg/dl)	.062	.383	.038	.643	.077	.726	**.923**	**0.003**
Fat mass (kg)	.029	.675	-.007	.928	.165	.430	.106	.822
Fat free mass (kg)	.013	.855	-.091	.259	.310	.132	.571	.181
Muscle mass (kg)	.030	.666	-.055	.488	.314	.126	.395	.380
TSH (mIU/l)	**.169**	**.015**	.142	.072	.081	.700	-.329	.388
Glucose (mg/dl)	-.004	.951	-.103	.199	.041	.846	.413	.269
HbA1c (%)	-.029	.680	-.052	.518	.056	.791	.446	.268
AST (IU/l)	-.024	.740	-.092	.261	**.431**	**.040**	.049	.909
ALT (IU/l)	.072	.312	.011	.896	**.537**	**.007**	.289	.487
Insulin (mIU/l)	.048	.526	.016	.857	.097	.692	.469	.288

BMI = body mass index, TG = triglyceride, TC = total cholesterol, LDL-C = low density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, TSH = thyroid stimulating hormone, T4 = thyroxin, HbA1c = glycated hemoglobin, ALT = Alanin transaminase, AST = Aspartat transaminase, HOMA-IR: Homeostatic Model of Assessment-Insulin Resistance

Table 3. The characteristics of the 78 patients at baseline and 6 months later

	Baseline (n = 78) Median (min-max)	6th months (n = 78) Median (min-max)	The difference between baseline and 6th month Median (min-max)
Age (years)	53.00 (21.00-78.00)	53.00 (21.00-78.00)	
Weight (kg)	96.95 (71.10-134.60)	90.70 (67.00-117.90)	-5.95 (-22.3—0.80)
BMI (kg/m²)	35.95 (31.20-50.00)	34.40 (28.10-42.30)	-2.00 (-8.2-0.10)
Fat mass (kg)	40.35 (23.10-60.20)	36.55 (20.90-50.90)	-3.50 (-12.50-0.90)
Fat-free mass (kg)	55.55 (47.40-81.50)	55.65 (45.30-78.70)	-1.30 (-4.20-2.20)
Muscle mass (kg)	52.00 (27.70-77.50)	51.75 (28.60-74.80)	-1.20 (-4.00-2.10)
TPOAb (IU/mL)	111.61 (7.92-1000)	84.5 (0.50-1000)	-25.41 (-283.74-5.45)
TSH (mIU/l)	2.37 (0.00-39.85)	-	

BMI = body mass index; TPO Ab =: Thyroid peroxidase antibody, TSH = thyroid stimulating hormone

body values decreased after weight loss ($p < 0.001$) (Fig. 2). When the effect of weight loss on TPO antibody in euthyroid, subclinical hypothyroid and overt hypothyroid groups were analyzed separately, the effect of weight loss was more evident in the euthyroid group than the hypothyroid group. The effect of weight loss wasn't observed in the overt hypothyroid group, however the number of the patients in this group was too small (n = 2) (Fig. 3).

The correlation between weight difference and TPO antibody difference was also investigated in the study. Weight and TPO antibody difference were calculated by subtracting the weight and TPO antibody values obtained in the second visit from the first visit. Visually such correlation can be seen from a scatter plot as depicted in Fig. 4. Fig. 3 implies that there is not relationship between difference in weight and difference in TPO antibody levels (r = 0.167, $p = 0.171$). Likewise, no significant correlation was found between the differences in fat mass and TPO antibody (r = 0.067, $p = 0.656$) (Fig. 5) and the muscle mass and TPO antibody values (r = -0.011, $p = 0.939$) at the end of the 6th month.

In our laboratory, antibody titers above 1000 IU/ml could not be calculated and were reported as > 1000 IU/ml. The values reported as > 1000 IU/ml were analyzed as 1000 IU/ml (n = 32). Therefore, the difference in TPO antibody values did not reflect the actual difference. This was the most important limitation of our study.

DISCUSSION

In this study it was observed that TPO antibody levels decreased after weight loss at the end of the 6th

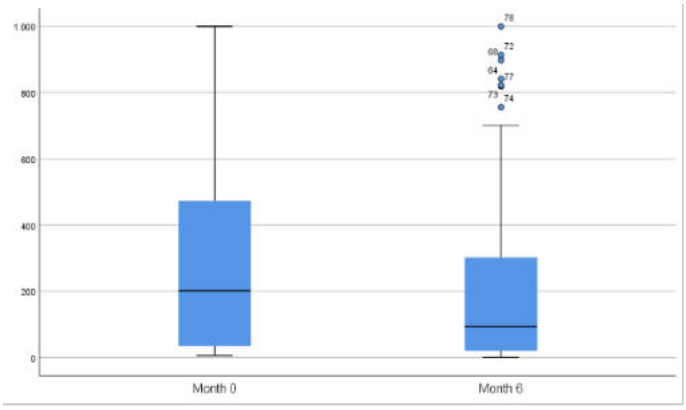

Fig. 2. The effect of weight loss on TPO antibody.

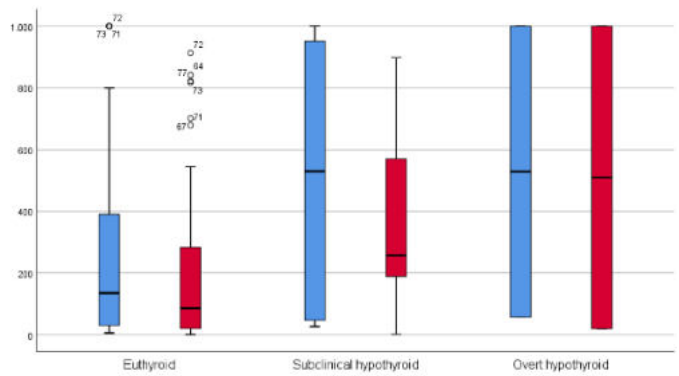

Fig. 3. The effect of weight loss on TPO antibody in euthyroid, subclinical hypothyroid and overt hypothyroid group.

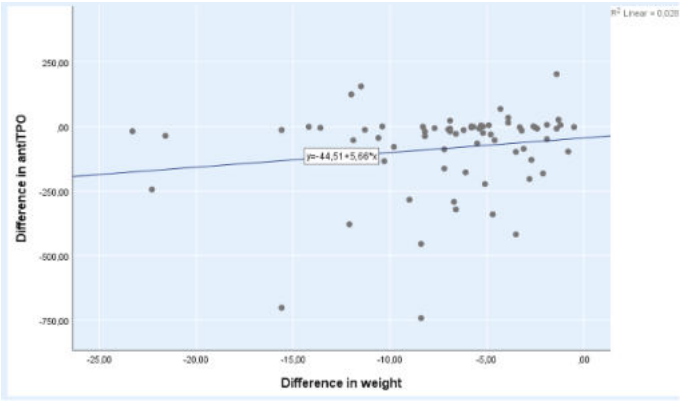

Fig. 4. The correlation between difference in weight and TPOAb difference in the 6th month.

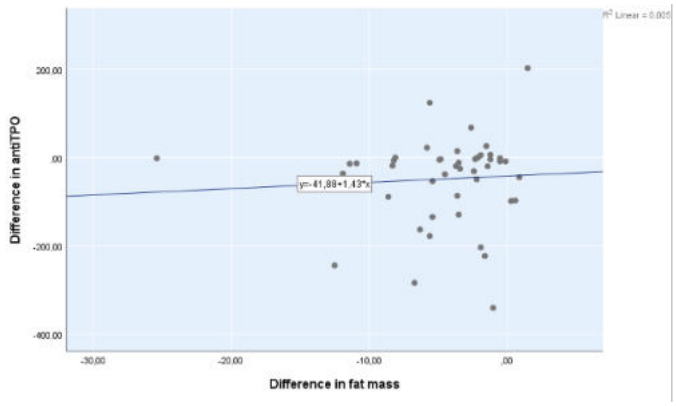

Fig. 5. Correlation between difference in fat mass and TPOAb in the 6th month.

month of follow-up and the effect of weight loss was more evident in the euthyroid group.

It has been demonstrated in a study that TPO antibody titers histopathologically show the degree of thyroid inflammation [6, 15]. In a study conducted by dividing euthyroid persons into two groups according to the presence of TPO antibody, higher TSH levels were shown in persons with a positive TPO antibody titer [7]. In a study including subclinical hypothyroidism, overt hypothyroidism and control groups; there was no significant difference between subclinical and overt hypothyroidism groups in terms of TPO antibody levels, while the lowest value was found in the control group [5]. In our study, TPO antibody levels were significantly higher in patients with overt hypothyroidism compared to those with subclinical hypothyroidism and in patients with subclinical hypothyroidism compared to the controls. There was a significant positive correlation between TPO antibody and TSH levels. Our findings supported the literature.

There are studies showing that the risk of thyroid autoimmunity is higher in persons with obesity and that being overweight in childhood increases the risk of development of thyroid autoimmunity between 60-64 years of age [16]. In addition, it was observed that thyroiditis is exaggerated as the level of obesity increases in patients with thyroid autoimmunity [17]. Studies investigating the relationship between BMI and TPO antibodies have reported different results. In the Danish National Birth Cohort study examining the correlation between BMI and the risk for development of autoimmune diseases; linear correlations were demonstrated between BMI and development of all autoimmune diseases except for ankylosing spondylitis, inflammatory bowel disease and sarcoidosis [14]. In a study from China, BMI was found to be correlated with TPO antibodies in women, while this correlation was not present in men. It has been reported that gender affects the correlation between obesity and autoimmune thyroiditis. This was thought to have resulted from the differences between women and men in terms of the distribution of body fat and secretion of adipokines and from the higher immune response in women [18, 19]. Unlike these studies, Knudsen *et al*. [20] could not detect a significant correlation between BMI and TPO antibody. In our study, there was a significant weak correlation between BMI and TPO antibody in patients with thyroid autoimmunity, but the significance was disappeared when the effect of TSH was considered.

In a study by Chen *et al*. [20], when adjusted for age, smoking, TSH, and menopause only in female patients, TPO antibody positivity was correlated with BMI, HbA1c, HDL, LDL, TC, TG, fasting plasma glucose (FPG) and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). The same correlation was not found when considering both genders and only male patients [20]. In our study, 91% of all participants were female and similarly to the above-mentioned study, no correlation was found between TPO antibody and these parameters when only female patients were considered. Again, no significant correlation was found between TPO antibody and these parameters when both genders were analyzed.

Studies have shown that obesity predisposes to many autoimmune diseases such as rheumatoid arthritis, multiple sclerosis, psoriasis, type 1 DM, inflam-

matory bowel disease and autoimmune thyroiditis and increased severity of these diseases [8, 10, 21]. On the other hand, studies about the effect of weight loss on autoimmune diseases are limited. In a review of many clinical trials, it was reported that psoriasis symptoms decreased in patients who lost weight and weight loss facilitated the treatment in these patients [22]. In a study conducted in patients with obesity with rheumatoid arthritis, a correlation was reported between BMI and number of the joints with arthritis and severity of the disease, and improvement in body composition should be a part of rheumatoid arthritis treatment [23]. Previous studies have only investigated the relationship between change of thyroid hormone levels with respect to body weight, ignoring the levels of TPO antibodies [24]. We could not find any study conducted about investigating the relationship between weight loss and TPO antibodies levels in thyroid autoimmunity in the literature. We consider this is the first study about this topic.

In a study searching the relationship between low-carbohydrate diet and thyroid autoimmunity, one group was given a diet rich in vegetables, protein, milk and dairy products, and poor in carbohydrates and the other group was given a reduced-calorie diet. In the carbohydrate-poor diet group a significant decrease in TPO antibodies was observed, while no change was detected in the reduced calorie diet group. However, those who were on a diet poor in carbohydrates had more weight loss. In our study, although the participants did not obey the same diet, weight loss led to decrease in TPO antibodies particularly in patients with euthyroid state [25].

Besides cytokines that have proinflammatory effect, adipokines which have anti-inflammatory effects are also secreted by the adipose tissue. Studies have shown that leptin which has proinflammatory effect was decreased and adipokines that have anti-inflammatory effects were increased in patients who lost weight after bariatric surgery [26-28] Other studies have reported that excessive inflammatory activity was decreased and anti-inflammatory activity was increased in persons who lost weight with diet and increased physical activity [26, 29]. In a study on mice, it was found that first immune response was increased with weight loss and then the immune response was decreased as weight loss was continued [30]. In a genetical study, it was shown that inflammation related gene expression in adipocytes and macrophages were modified and inflammatory response was decreased with weight loss [31]. This could explain our result as weight loss suppresses inflammation, and thus excessive immune response occurring against this, and accordingly provides a decrease in TPO antibodies.

In a study that compared the patients with and without obesity, weight loss was shown to decrease immune response in both groups, and this decrease was similar in these two groups [32]. In our study, all participants were affected with obesity, and therefore we could not perform this comparison, but we found that weight loss decreased TPO antibodies in persons with obesity and attributed this to the decreased immune response in these persons. On the other hand, to link the decrease in serum TPO antibody levels for six months, relatively a short observation period, to the reduced immune response, further controlled and long-term studies are needed.

Limitations

This study has several limitations. First, the number of participants was relatively small, and a large part of the participants lost from the follow up. However, due to the duration of the study, the high drop-out is expected, especially in a study examining an obesity outpatient clinic. Second, antibody titers above 1000 IU/ml could not be calculated in our laboratory and reported as >1000 IU/ml (n = 32). Therefore, difference in the TPO antibody levels could not be calculated in an accurately. Third, the study was conducted retrospectively and as a result it was not possible to take a control group. Forth, since this was a retrospective study the patients who had TPO antibody levels at baseline and 6th month were included in the study. The baseline TSH values were recorded from the patient's files however most of the patients' TSH levels could not be obtained from their files because TSH values of the patients were checked at various times other than the 6th month. Last, hence, mostly women refer to our obesity outpatient clinic, a high female/male ratio was detected in the study.

CONCLUSION

Today, the reason for the increase in autoimmune diseases is an issue of concern. Many factors have

been held responsible for this increase. Many studies have shown obesity as the most important factor for this increase. In our study, weight loss was found to decrease TPO antibody levels in persons with thyroid autıimmunity. We attributed this to decreased autoimmunity by weight loss in thyroid autıimmunity. However, further comprehensive and randomized-controlled studies are needed to confirm this.

Authors' Contribution

Study Conception: HHM, HHM; Study Design: HHM, HHM; Supervision: HHM, HHM; Funding: HHM; Materials: HHM; Data Collection and/or Processing: HHM, HHM; Statistical Analysis and/or Data Interpretation: HHM, HHM; Literature Review: HHM, HHM; Manuscript Preparation: HHM, HHM and Critical Review: HHM, HHM.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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