Thyroid Hormones and Thyroid Antibodies in Patients with Alopecia Areata

ALOPESİ AREATALI HASTALARDA TİROİD HORMONLARI VE TİROİD ANTİKORLARI

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Summary

Alopecia areata (AA) is a common disease of unknown etiology characterised by the appearance of patchy areas of hair loss leaving a smooth and non-scarred scalp . AA may be associated with autoimmune disorders, especially of the thyroid. The prevalence of thyroid disease in patient with AA previously reported varied from 0 to 28%.

Fonvpatients with alopecia areata were studied. A complete history was taken and physical examination was performed. Serum triiodothyronine, thyroxine, free thyroxine, free triiodothyronine, thyroid-stimulating hormone, trimicrosomal antibody and thyroglobulin antibody levels were measured in every patient. The control group consisted of 20, age and sex-matched healthy volunteers.

Among 40 patients, aged 6-65 years, six cases had non-toxic goitre. Except for triiodothyronine and free-lriidothyronine, there were not any significant differences between the patients and control groups in terms of thyroid parameters. Three patients (7.5 %) had positive microsomal antibodies.

We determined that there is an association between alopecia areata and thyroid diseases.

Key Words: Alopecia areata, Thyroid hormones, Thyroid antibodies

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Özet

Alopecia areata (AA) etiyolojisi bilinmeyen, skatrissiz yama tarzında kıl dökülmesi ile karekterize sık görülen bir hastalıktır. AA başta tiroid hastalıkları olmak üzere otoimmün hastalıklarla birlikte görülebilir. Daha önceki çalışmalarda AA'da tiroid hastalıkları görülme sıklığı % 0-28 olarak bildirilmiştir.

Bu çalışma 40 alopecia areatalı hastada yapıldı. Bütün hastaların anamnezleri alındı ve fizik muayeneleri yapıldı. Her hastadan serum triiyodotironin, tiroksin, serbest-tiroksin, serbest-triiyodotironin, tiroid stimülan hormon, trimikrozomal antikor ve tiroglobülin antikor seviyeleri ölçüldü. Yaş ve cinsiyetleri olgularımızla uyumlu 20 sağlıklı ve gönüllü kişi kontrol grubumuzu oluşturdu.

6-65 yaşları arasında olan 40 hastanın altısında noıı-toksik guatr tespit edildi. Serum triiyodotironin ve serbest-triiyodotironin haricindeki tiroid parametreleri bakımından hastalar ve kontrol grubu arasında önemli bir farklılık yoktu. Üç hastada (%7.5) trimikrosomal antikorlar pozitif bulundu.

Bu çalışmada tiroid hastalıkları ile alopecia areata arasında bir ilişki olduğunu belirledik.

Anahtar Kelimeler: Alopesia areata, Tiroid hormonları, Tiroid antikorları

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Alopecia areata (AA) is a common disease, with an incidence of 2 to 3% among the dermatoses and 0.1 % in the population at large (1,2). Although the etiology of AA still remains unknown, many theories have been proposed, such as the association of AA with trauma, psychosomatic factors, genetics, endocrine diseases, bacterial and viral infections, vascular insufficiency, atopic state and autoimmune diseases (1-4). Thyroid diseases were found to be associated with AA in approximately 0-28% of patients (3-6). The aim of this study was to determine the levels of thyroid hormones and thyroid antibodies in patients with A A .

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Materials and Methods

We have studied 40 patients with AA and 20 healthy controls who were age and sex-matched

volunteers. Among 40 patients with AA, 4 (10%) were females and 36 (90%) were males. The ages ranged from 6 to 65 years (mean 32.4 ± 8.32 years). Among 20 controls, 5 (25%) were females and 15 (75%) were males. The ages ranged from 10 to 62 years (mean 34.7 ± 7.43 years). All patients with AA attending the dermatology outpatient department from January 1997 to August 1997 were included in the study. All patients were submitted to a complete clinical and dermatological examination. All of our patients were consulted with an endocrinologist. Laboratory evaluations were conducted in every patient and control subjects including thyroid function tests such as triiodothyronine (T3), free triiodothyronine (FT3), thyroxine (T4), free thyroxine (FT4), thyroid-stimulating hormone (TSH), tnmicrosomal antibody (TmAb), and thyroglobulin antibody (TgAb).

After an overnight fasting, 8-10 ml blood was drawn into heparinized vacutainer glass tubes by venipuncture. Blood samples were centnfuged at 2000xg for 10 min and serum samples obtained were stored at -20 °C until assayed. Serum T3, T4, FT3, FT4 and TSH levels were determined by chelimuninesccnce method with commercially available kits (Chiron Diagnostics, USA). Serum TmAb and TgAb levels were measured with a commercially available kit (Thyroid Autoimmunity Test, Immuno Dot*).

The data were given mean ± standard deviation. Calculations of statistical significance were performed by Student's t test. A value lower than 0.05 was accepted as meaningful.

Results

There were non-toxic goitre in 6 patients (4 females and 2 males). These patients had elevated serum FT3 and T3. Twenty-six patients (65%) had only one area of hair loss. No alopecia totalis or universalis was detected in the patients. Except for serum T3 and FT3, there was no significant difference between the patient and control groups in terms of thyroid parameters (Table 1). Scrum FT3 levels of the patients ranged from 2 to 11 ng/ml (5.48 ±1.382 ng/ml) whereas all serum FT3 levels of the controls were within the normal ranges (2.1 to 3.6 ng/ml and mean: 2.80 =n 0.476 ng/ml) and the difference was statistically significant ($p<0.001$). Additionally, T3 levels of the patients ranged from 137 to 231 ng/ml (168.400*21.701 ng/ml) whereas all scrum T3 levels of controls were within the normal ranges (94 to 118 ng/ml and mean: 104.700.-9.417 ng/ml) and the difference was statistically significant ($p<0.001$). Three patients (7.5%) had positive microsomal antibodies. Although these patients had elevated serum FT3 and T3, they had no signs or symptoms of thyroid diseases. The prevalence of positive TmAb in the AA group was not statistically different from that of the control group ($p>0.05$).

Discussion

AA is known to be associated with certain organ-specific autoimmune diseases. The autoimmune diseases associated with AA include Hashimato's thyroiditis, Graves' disease, insiilinc-dependent diabetes mcllitus. vitiligo, Addison's disease (3). AA was found to be associated with various thyroid diseases (7,8). Unfortunately, pathogenic mechanism of that association is between AA and thyroid diseases not explained, yet (5.9,10). Morgans found that AA occurred more frequently in patients with thyrotoxicosis (9). Midler and Winkeimann determined Hashimato's disease in 8% of 736 patients of AA (10). Additionally, AA was found to be associated with Graves' disease, thyroid adenoma and simple goiter (3,5,6,9,11). There was non-toxic goiter in our six patients.

Table 1. Thyroid hormones activities in serum of patients with AA and controls

Parameters	AA patients (n: 40)	Controls (n: 20)	p value	Normal value
Triidothyroninc (ng/dl)	168.400±21.701	104.700±9.417	pO.OOl	60-180
Frce-triidothyronm-j (ng/dl)	5.480 ± 1.382	2.800 ± 0.476	p<0.001	2.3-4.2
Thyroxine (mg/dl)	8.1800 ± 1.429	8.460 ± 1.345	p>0.5	2-13
Frec-thyroxine (ng/dl)	1.100 ±0.149	1.140 ± 0.158	p>0.5	0.8-1.8
Thyroid-Stimulating hormone (mu/ml)	2.060 ± 0.938	1.550 ±0,272	p>0.5	0.4-5.0

The study performed by Cunliffe and colleagues showed an association between AA and thyroid disease. They determined thyroid disease (thyrotoxicosis, non-toxic goitre and Hashimato's disease) in 24% of their patients with AA. In addition, they determined that 2 males of 71 patients had positive thyroid antibodies (9). In the present study, we reported that 15% of the patients had thyroid disease (Non-toxic goitre); this percentage was lower than that of Cunliffe et all's study. We also found that 3 males of 40 cases had antithyroid antibodies, this percentage was three times higher than that of Cunliffe et all's study. Puavilai S and Puavilai G had detected microsomal antibodies in 4.6% of AA cases. This result was similar to our conclusion. On the other hand, there are various results in the literature. For example, Down and Kumar have determined thyroid disease in 1% of AA (5). De Wcert and colleagues, studied 100 patients, showed that there was antithyroid antibody positivity at 15% ratio (3). Dcrici and colleagues found thyroid disease (Thyrotoxicosis, thyroid adenoma, Hashimoto's disease and non-toxic goitre) in 10% of their patients with AA (11). Lenk and colleagues determined antithyroid antibodies in 14.6% of 41 patients with AA. Their patients had no symptom of thyroid diseases (12). Giilekon and colleagues did not find antithyroid antibodies in their AA patients (13).

There is evidence that immunologic processes play an important role in the pathogenesis of AA. In autoimmune polyendrocrinopathy, 30% of the patients develop AA, Hashimato's disease and myasthenia gravis has been repeatedly reported (9,14,15). Although there is an association between AA and endocrine diseases, etiologic and pathogenic mechanism of the association of AA with endocrine diseases is remains to be enlightened.

In conclusion, because the estimated prevalence of thyroid disease in our patients with AA was relatively high, we recommend that use of thyroid function tests, especially T3 and FT3, might be valuable in determining thyroid disease in patients with AA, even though signs or symptoms of thyroid diseases are not present.

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